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CONTRIBUTION À L'ÉTUDE DES TROUBLES DE LA MÉNOPAUSE ROENTGÉNIENNE

COMPARAISON AVEC LES TROUBLES DES MÉNOPAUSES CHIRURGICALES

par

R. Gilbert et *A. Eghiayan*
Médecin-Chef Premier-Assistant

La faveur accordée de plus en plus à la ménopause roentgénienne, dans le grand nombre des cas où une ménopause artificielle s'impose, a suscité des études nouvelles, et des comparaisons intéressantes avec les ménopauses chirurgicales.

La discussion porte entre autres sur la question suivante: quelles sont les différences, qualitatives et quantitatives, des *troubles des ménopauses* artificielles, selon que la ménopause est provoquée par les rayons Roentgen, par l'ablation des ovaires avec ou sans hystérectomie, ou par l'hystérectomie seule.

Bon nombre d'auteurs, en majorité des gynécologues de langue allemande, admettent que les troubles de la ménopause roentgénienne sont relativement moins fréquents et moins accusés, que ceux des ménopauses chirurgicales. Cela amène certains d'entre eux à penser qu'il existe probablement, dans l'ovaire de la femme, une glande à sécrétion interne individualisée qui, grâce à sa moindre radiosensibilité, serait plus ou moins épargnée par une dose de R R[1] capable de détruire, d'autre part, la totalité des follicules; dès lors, le terme de *castration roentgénienne* a été considéré comme impropre, et l'on a proposé de lui substituer, pour faire ressortir les caractères différentiels de la ménopause roentgénienne, des termes tels que: *exovulation*, *désovulation*, *ovonécrose* (SEITZ et WINTZ), *stérilisation roentgénienne* (G. SCHWARZ), *ménopause roentgénienne* (FUCHS) etc.

SEITZ et WINTZ, en se basant sur leur expérience clinique, ont même parlé d'une *castration graduelle;* il résulte de leurs constatations qu'il serait possible, dans une certaine mesure, et proportionnellement à la dose de R R employée, d'obtenir: soit la castration *temporaire*, par la

[1] Abrévation pour Rayons Roentgen.

destruction d'une partie des follicules (follicules mûrs); soit la castration *définitive* par la destruction de tous les follicules, tout en conservant la glande interstitielle; soit enfin la castration *totale*, par la destruction simultanée des follicules et de la glande interstitielle.

Il faut reconnaître cependant que, faute de preuves histologiques concluantes, chez la femme, les travaux nombreux et importants publiés sur ce sujet, n'ont pas mis un terme aux divergences profondes qui s'étaient manifestées.

La question, qui n'a pas seulement un intérêt théorique, mais un intérêt thérapeutique, reste ouverte et notre contribution a pour but d'ajouter aux faits connus de nouvelles observations.

Nous avons repris les travaux et statistiques relatifs à la question dans le domaine chirurgical et dans le domaine radiologique (rayons Roentgen) en laissant de côté, pour le moment du moins, la curiethérapie.[1] Quant à nos propres observations, elles se rapportent toutes à la ménopause roentgénienne.

Nous nous proposons d'exposer plus particulièrement les troubles suivants:[2]

I) Les troubles vasomoteurs
   a) Bouffées de chaleur (avec ou sans transpirations)
   b) Hypertension artérielle
II) L'adipose
III) Les troubles nerveux
   a) Céphalées
   b) Asthénie neuro-musculaire
IV) Les troubles psychiques, soit: anxiété, irritabilité, nervosisme, instabilité de caractère, dépression, mélancolie
V) La diminution de la mémoire
VI) Les troubles du sens génital
VII) Les accidents cutanés
   a) Dermatoses
   b) Prurit
   c) Chute des cheveux
VIII) La constipation.

Nos recherches portent sur *50 cas*.

Nous avons éliminé soit les cas trop récents, soit les cas trop anciens, soit les malades qui ne se sont pas présentées à nos convocations. Nous avons tenu en effet à contrôler les malades personnellement; nous nous

[1] Nous n'avons pas trouvé, dans le domaine de la curiethérapie, de travaux à mettre en parallèle avec ceux qui font l'objet de cette étude.

[2] Nous n'avons retenu pour le moment que les troubles présentant un certain intérêt, laissant de côté ceux qui se sont révélés d'importance très secondaire.

sommes refusé à nous contenter de renseignements reçus par correspondance. Enfin, nous avons éliminé les malades traitées à un âge trop avancé pour que l'on puisse juger de la part qui revenait à l'effet des rayons Roentgen.

Nos recherches, sur les troubles de la ménopause roentgénienne, ont commencé il y a quinze mois (mars 1927). Ce sont donc les cas postérieurs à cette date qui ont subi des contrôles minutieux et répétés, *avant et après* la castration.[1]

Les cas plus anciens, échelonnés sur les 3 à 4 années précédentes, n'ont été contrôlés qu'*après* la castration roentgénienne. On pourra objecter que des contrôles, faits plus ou moins longtemps après l'installation de l'aménorrhée, sont discutables. On sait, en effet, que les troubles de la castration ainsi que ceux qui accompagnent la ménopause naturelle, ne sont pas tous définitifs, mais disparaissent d'habitude, en partie, au bout d'un an ou deux, grâce à l'adaptation progressive de l'organisme aux conditions nouvelles. Cependant, étant donné que la majorité des troubles étudiés donnent des phénomènes subjectifs, nous avons pu, même dans les cas anciens où les troubles avaient complètement cessé[2] obtenir des renseignements précis et complets, grâce à un interrogatoire serré, et grâce surtout au fait qu'il s'agissait de personnes intelligentes et s'observant bien. Seuls manquent les renseignements relatifs à la pression sanguine *avant* la radiothérapie.

Voici, brièvement résumées,[3] quelques indications relatives à l'âge des malades, et au diagnostic de l'affection traitée.

Age:

Malades âgées de 26 à 30 ans	. . . . .	2 cas
» » » 31 » 35 »	. . . . .	4 »
» » » 36 » 40 »	. . . . .	5 »
» » » 41 » 45 »	. . . . .	16 »
» » » 46 » 50 »	. . . . .	18 »
» » » 51 » 55 »	. . . . .	5 »
	total	50 »

Diagnostic:

Fibro-myomes	. . . . .	39 cas
Métropathies hémorragiques	. . . . .	3 »
Troubles psychiques, hyperovarie	. . . . .	4 »

[1] Pour les derniers cas traités, l'intervalle minimum entre l'installation de l'aménorrhée et le dernier contrôle a été de 2 mois.

[2] Ces cas sont d'ailleurs une faible minorité dans notre statistique.

[3] Nous avions groupé en *tableaux synoptiques* une série de renseignements relatifs à chaque cas. Nous avons dû renoncer au dernier moment à publier ces tableaux, faute de place.

Foyers abdominaux de granulomatose maligne .	2 cas
Cancer utérin	1 »
Métastases osseuses d'un Ca du sein opéré . . .	1 »
total	50 »

Nous ne parlerons pas des résultats thérapeutiques (roentgenthérapie) ni des détails techniques de nos traitements, ces sujets ayant fait l'objet d'une publication antérieure de l'un de nous[1] pour les cas traités jusqu'au début de 1926. Rappelons seulement dans les grandes lignes, la technique que nous pratiquons depuis bientôt 6 ans et qui nous a toujours donné d'excellents résultats.

Technique radiothérapique. Irradiations par 4 champs, convergeant deux par deux de chaque côté de la ligne médiane (deux ventraux et deux dorsaux), toujours perpendiculaires au plan sur lequel est étendu à plat la malade, de telle sorte que les faisceaux frappent à la fois les régions ovariennes et l'utérus (particulièrement pour le cas des fibromyomes). Les dimensions de chaque porte d'entrée sont, selon les cas, de 100 à 150 cm² (et plus dans les cas de grosses tumeurs). Le localisateur comprime les plans superficiels dans la mesure du possible. La distance focale est de 30 à 40 cm, suivant le volume des tumeurs. Chaque champ est irradié deux fois et reçoit chaque fois une dose de 650 à 750 R (français)[2] à la peau pour les cas moyens; ainsi chacun des 4 champs reçoit au total 1300 à 1500 R. La durée de chaque séance varie selon l'appareil et selon la malade; pour 30 cm, de distance focale, de 8 à 15 minutes; cela fait au total une à deux heures d'irradiation étalées sur une dizaine de jours. Les appareils que nous utilisons le plus couramment dans ce genre de traitement sont: l'inducteur »Néo-Symétrie» (E E 41—42 cm pointe-plateau, et 2.2 m A) et la »Tension Constante» (200 Kv et 4 m A). Tubes Coolidge à anticathode de tungstène. Filtration: 5/10 mm *Zn* + 10/10 *Al*. D'après les mesures faites avec l'ionomètre de Solomon, le second modèle de chambre, et l'étalonneur Béclère-Solomon, le taux de transmission pour 150 cm² d'ouverture à la peau et 30 cm. de distance focale, à 10 cm. de profondeur, varie de 32 % à 35 % selon l'appareil.

Examen des troubles de ménopause artificielle. Comparaison des résultats chirurgicaux et radiologiques

Nous mettrons en parallèle la fréquence des troubles consécutifs aux ménopauses artificielles par:

[1] R. GILBERT: Un procédé de roentgenthérapie des fibromyomes utérins et des métropathies hémorragiques (*J. belge de radiologie*, 1926; et *J. de Radiologie et d'Electrologie*, Paris 1927, p. 282).

[2] *Note au moment des corrections:* L'un de nous a étalonné récemment au *Kustner*, dans le service de M. le Dr GUNSETT à Strasbourg, l'ionomètre de Solomon utilisé pour ces mesures. Le rapport entre l'R français et l'*r* international (après correction de 6,6 %), toutes conditions égales, a été trouvé égal à 2,77: 1. Cela donne, pour 1 300 à 1 500 R: *470* à *540 r*.

1) Ablation des ovaires seuls
2) Ablation des ovaires avec hystérectomie
3) Hystérectomie seule
4) Roentgenthérapie.

C'est dans ce dernier groupe que rentrent nos observations personnelles. Nos chiffres statistiques feront suite à ceux des auteurs de cette 4e catégorie.[1]

I. Troubles circulatoires.

a) *Bouffées de chaleur* (avec ou sans transpirations).

Nous n'avons pas adopté la classification de ces troubles en 3 ou 4 degrés d'intensité, ainsi que l'ont proposé certains auteurs. Ces distinctions ne nous ont pas paru nécessaires, du moins chez nos malades, car l'influence des facteurs psychiques, de l'hygiène générale, de la température ambiante, nous a paru jouer un rôle important dans ces troubles.

Nous pouvons affirmer que nos malades, à une ou deux exceptions près, n'ont jamais formulé de plaintes sur l'intensité des bouffées de chaleur ou des transpirations; et malgré notre insistance, elles nous ont déclaré catégoriquement que ces troubles sont peu gênants, très supportables, lors même que dans la grande majorité des cas, ils étaient quotidiens.

Il est vrai qu'ils sont d'autant plus facilement acceptés que leur apparition coïncide à peu près avec la disparition des troubles, autrement gênants ou sérieux, qui avaient motivé le traitement. Souvent aussi nous avons entendu dire par nos malades que les bouffées de chaleur sont si insignifiantes que »ce n'est vraiment pas la peine d'en parler».

Bouffées de chaleur (avec ou sans transpirations)

Ablation des ovaires

			%
Canu	102 cas		76.47
		légères 5 %	
		moyennes 32.5 %	
		fortes 62.5 %	
Glaeveckf	42 cas		78.5
Jayle	40 »		97.5
Julien			86
Liesau	50 cas		90
Martin, L. F. A.	23 »		87
Pinesse	106 »		30

2) *Ablation des ovaires avec hystérectomie*

Mandl-Bürger	

[1] Nous avons tenu compte exclusivement des cas où l'aménorrhée complète a été obtenue, quel qu'ait été le mode de traitement.

		%
GÀL	51 cas (hystérectomie tot. ou supravagin.) . . .	82.35
GRAVES	233 »	80
JAYLE	14 »	100
TAUFFER	(cité par GÀL)	65.8
WERTH	(d°)	87
FERRY	50 cas (hystérectomie totale, sauf 4 qui ont conservé leur utérus)	96

3) *Hystérectomie.*

ALTERTHUM	(supravaginale)	40
GEMACH—GAUSS	(cité par FUCHS) (totale)	69
GÀL	21 cas (totale ou supravaginale)	33.33
GLAEVECKE	12 » (totale)	33.33
GNAU—GAUSS	(cité par FUCHS) (totale)	56
GRAVES	26 cas (totale)	81
JAYLE	15 » (totale)	53
MANDL—BÜRGER	. . . . .	67.7
TAUFFER (totale)	. . . . .	49

4) *Roentgenthérapie.*

BRETSCHNEIDER	43 cas (bouf. de chal., transpir. et palpitations)	48.84
	faibles 42.66 %	
	moyennes 33.33 %	
	fortes 24 %	
FELDWEG	205 cas (bouf. de chal., transpir., vertiges, palpitations)	90.6
FUCHS	69 »	91.2
	légères 30.2 %	
	moyennes 46 %	
	très fortes 23.8 %	
GÀL	115 cas	80.9
	légères 34.73 %	
	moyennes 50.52 %	
	très fortes 14.75 %	
GUTTMANN et BOTT	89 cas (castration temporaire)	84.7
	légères 25.9 %	
	moyennes 55.3 %	
	très fortes 3.5 %[1]	
R. SCHMID	601 cas (troubles vaso-moteurs)	(?) 11.83
Notre statistique	50 »	88

Examinons les pourcentages donnés par les statistiques des groupes 1, 2 et 4; sauf quelques exceptions, à l'avantage ou en défaveur de tel ou tel procédé, les chiffres se tiennent en général de près (ceux de la Roentgenthérapie sont les plus bas). L'impression qui s'en dégage est que *les bouffées de chaleur*, avec ou sans transpirations, ont une grande

[1] Il s'agit d'après les auteurs de femmes déjà antérieurement très nerveuses et psycholabiles.

fréquence et varient dans des limites relativement restreintes, après la *ménopause artificielle*, que *celle-ci soit provoquée par l'ablation des ovaires seuls*, par l'*ablation des ovaires* et *de l'utérus* (totale ou supravaginale) ou par la *roentgenthérapie*. Par contre, ce trouble se révèle moins fréquent après l'hystérectomie.

Mentionnons en passant, les résultats de notre enquête au point de vue *vertiges*, *palpitations* et *bourdonnement d'oreilles:*

Dans aucun de nos 50 cas nous n'avons pu enregistrer de tels symptômes, comme suites de la castration roentgénienne. Tout au contraire, bon nombre de nos patientes, qui en souffraient auparavant, nous ont affirmé qu'elles en ont été délivrées, après l'établissement de l'aménorrhée. Cela s'explique aisément, si l'on songe que ces troubles étaient liés à l'anémie provoquée par les hémorragies; l'aménorrhée, en permettant la restauration sanguine, restitue assez promptement à ces malades un meilleur état général et le relèvement de leurs forces.

b) *Hypertension*

Malgré un grand nombre de travaux, la question de l'*hypertension de la ménopause* en général, est loin d'être tranchée.

Nous voyons, d'un côté, les auteurs qui nient la réalité d'une telle hypertension (MOSBACHER et MEYER, O. SCHLESINGER, F. KISCH, H. WOLLSTEIN, J. WIESEL, L. KRAUL, H. LEHFELD, etc.). Ils se basent sur les résultats de recherches cliniques fort nombreuses, comportant des cas de ménopause naturelle, physiologique ou pathologique, et de ménopause artificielle, chirurgicale ou roentgénienne.

Dans les cas où une hypertension a été décelée, l'examen du sujet semble avoir démontré qu'il s'agissait d'une cause toujours autre que la suppression des règles ou de la fonction ovarienne. Il ressort cependant, de la plupart des recherches des auteurs cités, que la tension artérielle peut devenir très instable à la suite de la ménopause, subir des oscillations en plus ou en moins, de façon passagère, pouvant faire croire à une hypertension réelle, suivant les cas, si les examens sont peu répétés.

D'un autre côté, des recherches cliniques, également nombreuses, semblent démontrer qu'il existe bel et bien une hypertension de la ménopause, en l'absence de toute cause apparente, hypertension coïncidant avec l'établissement de l'aménorrhée (SCHICKELE, ASCHNER, F. HAUB, F. KULBS, P. DALEAS, F. GROEDEL, H. PAILLARD, LECONTE, etc.).

En outre, certaines recherches expérimentales (notamment celles de CHAMPY et GLEY), sur lesquelles nous n'avons pas à insister ici, semblent prouver l'action hypotensive des ovaires.

Entre ces deux opinions extrêmes, il existe cependant des opinions intermédiaires; ces auteurs, tout en admettant l'élévation de la tension artérielle, à la suite de la ménopause, font remarquer qu'elle frappe

seules les personnes qui ont eu déjà une tendance à l'hypertension *avant* la ménopause (G. COTTE); ou encore ils font observer que la ménopause peut amener l'hypotension, au même titre que l'hypertension.

Enfin, E. STRASSMANN est arrivé, par ses recherches, à la conclusion que la castration chirurgicale augmente régulièrement la pression systolique (en moyenne de 32 mm Hg) et la pression diastolique (de 15 mm Hg), mais seulement chez les femmes réglées au moment de l'opération, et non chez celles qui avaient atteint la ménopause naturelle. De plus, STRASSMANN conclut que la *castration roentgénienne* n'influence en aucun cas la pression sanguine si l'on a appliqué la »*dose ovarienne*»; par contre, en cas d'irradiations à doses massives, la pression augmente au même titre qu'en cas de castration chirurgicale.

Nos recherches systématiques comportent jusqu'à maintenant 18 cas (en majeure partie de fibro-myomes) chez des femmes n'ayant pas atteint la ménopause naturelle. L'âge des malades variait de 35 à 52 ans. Nous avons mesuré la pression artérielle à plusieurs reprises, chez chaque malade, *avant*, *pendant* et *après* le traitement, ainsi qu'après l'établissement de l'aménorrhée complète. L'intervalle entre l'installation de l'aménorrhée et la dernière mesure varie de 2 à 12 mois, suivant les patientes.

Comme appareil de mesure, nous nous servons soit de l'oscillomètre de Pachon, dont les limites normales habituelles sont: max. 140—160 mm et min. 80—100 mm Hg; soit du sphygmotensiomètre de Vaquez-Laubry, dont les chiffres respectifs sont de 10 à 30 mm inférieurs aux précédents.

Toutes les précautions habituelles ont été prises, afin que les mesures soient effectuées dans des conditions comparables et que les causes d'erreurs soient aussi restreintes que possible. En particulier, nous mettons le sujet au repos absolu, en décubitus dorsal, pendant les 10 minutes qui précèdent la mesure de la pression.

Voici les résultats trouvés chez les 18 malades observées systématiquement:

dans 5 cas aucune modification;

» 12 » des variations en plus ou en moins, allant de 5 à 15 mm, sans que les limites physiologiques maxima soient dépassées;

» 1 » (N° 11) la pression, qui était 21/10 avant l'aménorrhée, est tombée et s'est maintenue à 18/10. Cette baisse est attribuable à la cessation de l'abus de café que la malade a consentie sur notre conseil.

Chez les 32 autres malades, nous n'avons pu mesurer la pression sanguine qu'après l'aménorrhée nous préférons donc ne pas en tenir

compte, étant donné que chez bon nombre de ces malades, l'intervalle entre l'aménorrhée et la mesure de la pression était trop grand.

En résumé, *l'influence de la ménopause roentgénienne, sur la pression artérielle, dans les 18 cas que nous avons observés méthodiquement, paraît négligeable.*

Peut-on porter un jugement plus général sur la question de l'hypertension de la ménopause à l'heure actuelle? Nous ne le croyons pas.

De nouvelles recherches, basées sur un plus grand nombre de cas, et tenant compte de multiples causes d'erreurs, nous paraissent nécessaires. Elles doivent se baser, à notre avis, non seulement sur des contrôles répétés de la pression artérielle, avant et après la ménopause, mais aussi sur une exploration générale, clinique et radiologique, du système circulatoire et rénal, dans le but de dépister les stigmates d'une hypertension qui peut exister avant l'aménorrhée, mais qui est peut-être masquée au moment des mesures de la pression artérielle, par suite d'hémorragies abondantes.

II. Adipose

Il est évident que toute augmentation de poids, survenant à la suite de la ménopause artificielle, ne pourrait être considérée comme la conséquence pathologique de celle-ci. En effet, nombreuses sont les femmes, atteintes de fibro-myome, de métropathies hémorragiques, d'annexites et *a fortiori*, de cancer gynécologique, qui arrivent chez le médecin dans un état d'amaigrissement plus ou moins prononcé par suite de leur affection. Que ces femmes reviennent à leur poids normal, à la suite du traitement, cela est bien naturel, et ne peut être considéré comme un trouble de métabolisme.

Pour évaluer la part qui revient à ce trouble chez certaines femmes, on ne peut pas se baser uniquement sur l'augmentation de poids enregistrée, mais aussi sur les rapports entre le poids et la taille, et surtout entre le poids actuel et le poids habituel avant la maladie. Il importe, en outre, de tenir compte du type d'engraissement, à savoir s'il est général, uniforme, ou s'il intéresse plus spécialement certaines régions, notamment le bas-ventre et la racine des cuisses. C'est d'après ces principes que nous nous sommes guidés, dans l'appréciation de nos cas.

1) *Ablation des ovaires*

		%
Alterthum	182 cas	29.5
Glaevecke	40 »	57.5
Hagedorn	28 »	50
Jayle	40 »	17.5
Julien	28 »	60
Liesau	50 »	70

		%
L. F. A. Martin	23 »	21.7
Mandl—Bürger		58.3
Pfister	116 cas	52
2) *Ablation des ovaires avec hystérectomie*		
Jayle	13 cas	38.5
3) *Hystérectomie*		
Glaevecke	12 cas	3.33
Jayle	12 »	42
Werth		19.6
Werth et Burger		53.7
4) *Roentgenthérapie*		
Bretschneider	43 cas	4.65
Fuchs	67 »	53.7
Feldweg	205 »	7.8
Gàl	115 »	9.5
Guttmann et Bott	89 »	6.74
Notre statistique	50 »	20

Dans les statistiques du dernier groupe, on le voit, le pourcentage des cas d'engraissement ne dépasse pas 20 %, sauf dans celle de Fuchs, où nous trouvons exceptionnellement le chiffre élevé de 53.7 %. Cependant, le tableau change du tout au tout, quand on analyse ce dernier chiffre, en se guidant d'après les commentaires de l'auteur même. Tout d'abord, dans les 28.3 % des cas, l'engraissement a consisté en une augmentation de poids, ne dépassant pas 5 kg, et portant sur une période de un à deux ans. Or, comme il s'agit généralement de malades amaigries antérieurement, à la suite d'hémorragies et d'autres troubles, une pareille augmentation de poids n'implique pas nécessairement l'existence de trouble de métabolisme, imputable à la castration roentgénienne. Il est même permis de la considérer plutôt comme une conséquence favorable de la suppression du mal. Reste le 25.3 % des cas, soit 17 malades, chez qui l'augmentation du poids a dépassé 5 kg. D'après Fuchs, 4 d'entre elles n'ont fait que récupérer leur poids habituel, antérieur à la maladie, sans qu'il en résulte une augmentation anormale de pannicule adipeux. Donc en réalité, le pourcentage des cas d'adipose dans la statistique de Fuchs se réduit à 19.4 % (13 cas). Selon l'auteur, il s'agissait le plus souvent de personnes ayant eu déjà antérieurement une tendance marquée à l'adipose endogène. »De sorte que», conclut Fuchs, »les cas d'engraissement imputable aux troubles de nutrition ovariprive, sont en faible minorité.»

Qu'est-ce que nous montrent les statistiques opératoires? Qu'il s'agisse d'ablation des ovaires, avec ou sans hystérectomie, ou qu'il

s'agisse d'hystérectomie simple, la fréquence d'engraissement est sensiblement plus grande qu'à la suite de la castration roentgénienne, puisqu'elle oscille, suivant les auteurs, entre 17.5 et 50 % et plus.

III. Troubles nerveux

a) *Céphalées* b) *Asthénie neuro-musculaire*

Divers auteurs ont insisté sur l'apparition de *céphalées* plus ou moins intenses à la suite de la castration chirurgicale, ou sur l'exagération de ce symptôme dans les cas où il existait avant l'opération. Il en est de même de l'*asthénie neuro-musculaire* caractérisée par une faiblesse, un abattement général, que rien n'explique, et qui, par exclusion de toute autre cause, semble être consécutive à la suppression brusque et précoce des fonctions ovariennes.

Etant donné l'importance de ces troubles, au point de vue social surtout, nous avons interrogé toutes nos malades à ce sujet. Quatre nous ont déclaré que les céphalées existaient avant la roentgenthérapie, et sont devenues plus fréquentes et plus intenses depuis l'aménorrhée. En revanche, plusieurs de nos malades se félicitaient d'être allégées, voir même complètement délivrées de céphalées plus ou moins pénibles, dont elles souffraient auparavant, parfois depuis de longues années.

En ce qui concerne l'*asthénie neuro-musculaire*, non seulement nous ne l'avons jamais rencontrée chez nos malades, mais presque toutes nous ont déclaré avec satisfaction que depuis l'aménorrhée roentgénienne, elles sentent une reprise de forces et d'énergie physiques, qu'elles ne connaissaient plus depuis longtemps.

1) *Ablation des ovaires*

		Céphalées %	*Asthénie n-m* %
Alterthum	65 cas	24.6	—
Canu	102 »	69.6	80.4 [1]
Ferry	51 »	44	57 [2]
Jayle	26 »	31	31
Julien	28 »	50	50
Liesau	50 »	16	10
Martin, L. F. Albert	23 »	48	—
Pinesse	106 »	14	6

2) *Ablation des ovaires avec hystérectomie*

Jayle	13 cas	46	46

[1] Très intense dans environ la moitié des cas, d'après les indications de l'auteur.

[2] D'après l'auteur, quelques malades étaient à tel point fatiguées, qu'elles étaient réduites à l'inaction pendant plusieurs mois, alors même qu'elles étaient parfaitement guéries et en bon état général.

3) *Hystérectomie.*

		Céphalées	*Asthénie n-m*
		%	%
JAYLE	14 cas	14	7

4) *Roentgenthérapie*

FELDWEG	205 cas	14	4.87
Notre statistique	50 »	8	0

Voici donc deux sortes de troubles nerveux, assez importants, qui, sans être constants, apparaissent tout de même avec un pourcentage relativement élevé, chez les malades qui ont subi l'ablation des ovaires avec ou sans hystérectomie, ou l'hystérectomie seule (moins fréquemment, il est vrai, après celle-ci). Leur absence, ou leur très faible fréquence, après la ménopause roentgénienne, est très remarquable.

Remarquons en passant que nous avons encore cherché, chez nos malades, l'existence éventuelle de *névralgies, de troubles de la phonation, de la vue et de l'ouïe*, consécutifs à la castration roentgénienne, sans en observer cependant un seul cas!

IV. Troubles psychiques

V. Diminution de la mémoire

Les *troubles psychiques* que l'on peut observer à la suite de la ménopause, naturelle ou artificielle, sont l'anxiété, l'irritabilité, le nervosisme, l'instabilité de caractère, la dépression, voire même la mélancolie.

Ces troubles peuvent apparaître d'emblée chez des sujets jusque-là indemnes, ou bien, ce qui est plus souvent le cas, s'exagérer à la suite de la ménopause chez des sujets qui en étaient affectés auparavant.

Dans certains cas cependant, on peut observer le phénomène inverse, c. à d. la disparition, à la suite de la ménopause, de troubles psychiques plus ou moins accusés.

La *diminution de la mémoire* intéresse le plus souvent les faits récents ou les faits à accomplir; ainsi par exemple, le sujet entre dans une chambre, dans le but de chercher un objet, mais au même moment, il oublie son but, ne se rappelle plus quel objet il cherche.

L'appréciation de la part qui revient à la ménopause, dans les modifications psychiques signalées, présente des difficultés qui ne sont pas insurmontables, lorsqu'il s'agit de personnes pourvues d'une certaine intelligence et d'esprit d'auto-inspection.

Il va de soi qu'il faut éliminer les causes déterminantes occasionnelles, d'ordre matériel, social ou moral, et celles qui sont inhérentes au progrès de l'âge; il faut enfin et surtout, préciser le rapport chronologique de

l'apparition des modifications psychiques, avec le moment de l'établissement de l'aménorrhée et les divers accidents qui l'accompagnent.

1) *Ablation des ovaires*

		Troubles psychiques %		*Diminution mémoire* %
Alterthum	111 cas	13.5		—
Canu	102 »	81.4[1]		—
Ferry	51 »	69		70[2]
Glaevecke	33 »	68		—
Hagedorn	32 »	18.7		37.5
Jayle	32 »	80	(42 cas)	78.50
Julien	28 »	50		40
Mandl—Bürger	309 »	41.6		30
Martin, L. F. Albert	23 »	48		30.43
Liesau	50 »	18		24
Pfister	110 »	45.5		68.5
Pinesse	106 »	30		—

2) *Ablation des ovaires avec hystérectomie*

Jayle	14 cas	73		100
Tauffer		12.5		

3) *Hystérectomie*

Glaevecke	13 cas (totale)	27		—
Jayle	14 »	7.		27
Mandl—Bürger	96 »	34.2	(69 cas)	35.4
Werth	51 »	31.7	(70 »)	44.3

4) *Roentgenthérapie*

Brohl—Gauss		13.6		—
Feldweg	205 cas	12.19		0.97
Fuchs	68 »	10.10		35.2[3]
Gál	115 »	15		—
Stammen—Gauss		14		—
Notre statistique	50 cas	10		14

En prenant la moyenne des chiffres de chacun de ces quatre groupes, on ne peut pas s'empêcher de conclure que les troubles psychiques et la diminution de la mémoire sont nettement moins fréquents à la suite de la ménopause roentgénienne qu'à la suite de l'ablation des ovaires,

[1] Très intenses dans environ 35 % des cas suivant les indications de l'auteur.

[2] L'auteur note que »c'est un des troubles les plus gênants et dont se plaignent le plus les malades».

[3] L'auteur insiste sur le fait que 4 femmes seulement étaient au-dessous de 35 ans; tandis que la plupart se trouvaient dans l'âge préménopausique.

avec ou sans hystérectomie. Quant à l'hystérectomie seule, elle tient à peu près le milieu entre la ménopause roentgénienne et les deux autres procédés, pour les troubles en question.

V. Troubles du sens génital

Les troubles du sens génital que l'on rencontre le plus souvent à la suite de la ménopause, portent sur le libido et la volupté. Ces sensations peuvent diminuer, voire même disparaître, ou au contraire s'exagérer, ce qui est relativement rare. Dans certains cas cependant, on constate une dissociation entre les deux phénomènes: la volupté persiste ou augmente même, alors que le désir subit une diminution plus ou moins marquée. Il convient donc de les étudier séparément, comme le font beaucoup d'auteurs.

L'étude des troubles sexuels est certainement la plus délicate, car aux divers facteurs qui compliquent l'appréciation exacte de la part qui revient à la ménopause, dans l'ensemble des troubles envisagés, viennent s'ajouter ici les sentiments de pudeur du sujet. C'est pourquoi dans plusieurs de nos cas, nous avons spontanément éliminé ces questions de l'interrogatoire. Nous les avons posées néanmoins dans la majorité des cas, après avoir fait comprendre l'intérêt scientifique de l'enquête, et nous avons obtenu sans peine les renseignements nécessaires. Nous avons même interrogé des célibataires en nous en tenant à la question du »libido».

		Libido—Voluptas (diminués ou supprimés)	
		%	%
1) *Ablation des ovaires*			
Alterthum	38 cas	68.40	68.60
Canu	102 »	49	—
Ferry	51 »	42	43[1]
Glaevecke	27 »	78	69
Hagedorn	13 »	54	77
Jayle	35 »	36	27[1]
Julien	28 »	43	43
Liesau	17 »	82	60
Pfister	99 »	59.60	60.60
2) *Ablation des ovaires et de l'utérus*			
Jayle	11 cas	36	10
3) *Hystérectomie*			
Glaevecke	10 cas	40	50
Jayle	12 »	27	24[1]

[1] D'après ces auteurs, la diminution ou la disparition du *libido* est due en partie aux rapports douloureux, conséquence de la castration.

	Libido—Voluptas (*diminués ou supprimés*)	
	%	%
WERTH	22	—
MANDL—BÜRGER	13.90	—

4) *Roentgenthérapie*

FELDWEG	159 cas	33.33	
FUCHS	56 »	21.40	
GUTTMANN et BOTT	89 »	29.40	
Notre statistique	42 »	50	—
» »	35 »	—	45.7

Les statistiques ci-dessus montrent en moyenne que c'est à la suite de l'ablation des ovaires seuls qu'on enregistre la plus forte diminution du libido, les autres interventions chirurgicales donnent des chiffres voisins de ceux de la roentgenthérapie.

Il en est de même de la sensation de volupté. Mais ici nous ne pouvons nous baser, au point de vue radiologique, que sur notre statistique, les autres auteurs n'ayant pas traité cette question séparément.

Un fait est important à souligner ici: les rapports sexuels deviennent douloureux dans un certain pourcentage des cas à la suite de l'intervention chirurgicale (FERRY, JAYLE), d'après MANDL-BÜRGER, dans 21.9 % des cas et d'après CANU dans 31.4 % des cas[1] soit par l'atrophie prononcée, soit par la déformation opératoire du tractus génital. Par contre, les rapports douloureux sont relativement rares, à notre connaissance, à la suite de la roentgenthérapie (d'après FUCHS, deux fois sur 69 cas, soit env. dans 3 % des cas).

En tout cas, le résultat de notre enquête sur ce point a été tout à fait négatif.

VI. Accidents cutanés

Certains accidents cutanés, par leur apparition inopinée coïncidant avec la ménopause, ainsi que par l'absence de leurs causes habituelles, semblent avoir été attribués, à juste titre, aux conséquences de la ménopause.

Nous ne discuterons pas ici le mécanisme de production de ces accidents, cela sort du cadre de ce travail. Disons seulement en passant qu'on a invoqué soit une origine endocrinienne ou hypersympathicotonique, soit une origine toxique, en rapport avec la rétention des menstrues.

[1] En outre, d'après le même auteur, les rapports sont devenus même impossibles ou presque, dans 25.7 % des cas.

La ménopause artificielle, comme la ménopause naturelle, peut donc s'accompagner d'accidents cutanés, qui sont principalement:

a) *Des dermatoses*, telles que l'urticaire, l'eczéma, la couperose.

b) *Le prurit*, en tant que symptôme primitif, dit »prurit essentiel». Ce prurit est un des plus rebelles à toute thérapeutique et peut intéresser surtout les parties génitales, la poitrine et les extrémités, quelquefois de façon assez intense pour dégénérer en une véritable obsession. On conçoit, d'autre part, que suivant sa localisation, il peut être très gênant, faire croire à de l'onanisme ou le susciter.

c) *La chute des cheveux* est heureusement passagère, et généralement pas très intense; les malades ne s'en aperçoivent d'habitude qu'en se coiffant.[1]

d) *L'hypertrichose*, notamment de la face.

e) *Les troubles de la pigmentation*.

Nous nous occuperons plus spécialement des trois premiers, en nous basant malheureusement sur une documentation assez restreinte.

GÀL a rencontré *le prurit* dans 14 % des cas chez 76 opérées, avec ou sans conservation des ovaires.

FERRY, dans 50 cas d'ablation des ovaires, note 16 % de *prurit* et 56 % de *chute des cheveux*.

CANU, dans 102 cas d'ablation des ovaires, a enregistré 23.5 % de *prurit*, très intense dans un tiers des cas.

Enfin dans 23 cas d'ablation totale utéro-ovarienne, L. F. ALBERT MARTIN donne pour le prurit le chiffre exceptionnellement bas de 4.35 %.

En ce qui concerne les *dermatoses*, nous les trouvons dans le travail de FERRY avec une fréquence de 4 %, dans 50 cas d'ablation des ovaires (plaques érythémateuses, sclérodermie); et dans celui de JAYLE avec une fréquence de 9 % (urticaire, furoncles), dans 33 cas d'ablation des ovaires et de 30 % (acné, herpès, purpura), dans 14 cas d'ablation totale utéro-ovarienne.

A la suite de la roentgenthérapie, nous trouvons comme fréquence des cas de *prurit:*

4.3 % dans les 115 cas de GÀL
2.4 % » » 205 » » FELDWEG
8 % » nos 50 » [2]

et comme fréquence des dermatoses:
0.97 % dans les 205 cas de FELDWEG (eczéma)
4. % » nos 50 » (éruption eczématiforme).

[1] FERRY, se basant sur des cas de castration chirurgicale, estime même que la chute des cheveux peut aboutir quelquefois à la calvitie.

[2] Dans la moitié des cas, le prurit existait auparavant et n'a fait qu'augmenter à la suite de la ménopause roentgénienne.

En ce qui concerne la *chute des cheveux*, nous l'avons rencontrée dans 16 % de nos 50 cas (nous n'avons pas eu sous la main d'autres statistiques radiologiques sur cette question). Nous ferons remarquer que cet incident a été de courte durée chez nos malades, sans dépasser quelques semaines, et qu'il a été peu marqué; c'est en se coiffant que nos patientes se sont aperçues qu'elles perdaient leurs cheveux plus que d'habitude. Il est à signaler d'autre part que dans plusieurs cas, les cheveux sont devenus au contraire plus fermes et plus solides, au lendemain de l'aménorrhée roentgénienne. Sans doute la suppression des hémorragies et le relèvement consécutif des forces de l'organisme en général sont la raison de ce changement favorable.

En résumé, à l'exception des résultats de MARTIN, tous les autres résultats opératoires dépassent de loin comme fréquence, ceux de la castration roentgénienne, notamment en ce qui concerne la *chute des cheveux*. Il nous paraît donc justifié de faire une distinction nette, au point de vue des accidents cutanés en général, entre la castration roentgénienne d'une part, et les diverses méthodes de ménopause chirurgicale d'autre part.

Notons en passant que nos recherches sur la *pigmentation* et l'*hypertrichose* sont restées tout à fait négatives dans les 50 cas.

VII. Constipation

D'après ADLER, la constipation survenant à la suite de la ménopause serait due à une hypersympathicotonie d'origine ovariprive. L'idée d'une origine ovarienne semble être justifiée, en outre, par le fait que cette constipation survenue à la suite de l'aménorrhée, est bien influencée par l'opothérapie ovarienne (GÀL) tandis qu'elle se montre généralement très rebelle aux purgatifs.

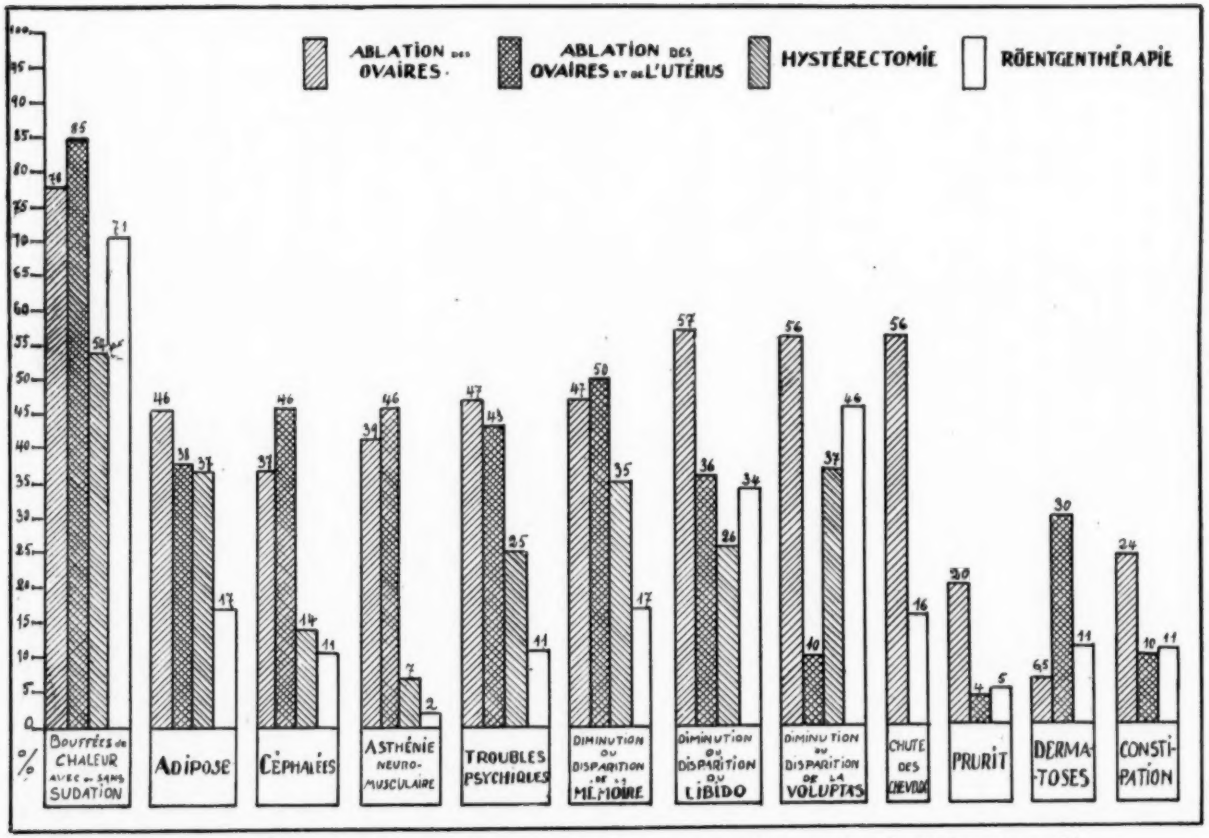
Dans ce domaine encore, nous n'avons pas pu trouver une documentation aussi abondante que nous l'aurions souhaité.

GÀL sur 76 opérées avec ou sans conservation des ovaires, a enregistré la constipation dans 14 % des cas, contre 4.3 % chez 115 malades ayant subi la castration roentgénienne.

CANU donne un chiffre beaucoup plus élevé: 65.6 % dans 102 cas d'ablation des ovaires. Dans 44.7 % des constipées, ce trouble était très prononcé.

Par contre, JAYLE, et L. F. ALBERT MARTIN, donnent des chiffres très bas dans des cas d'ablation des ovaires; soit respectivement: 3 % dans 33 cas et 4.35 % dans 23 cas.

D'un autre côté, JAYLE note 10 % de constipées, sur 14 femmes ayant subi l'ablation des ovaires et de l'utérus.



Fréquence des troubles consécutifs aux ménopauses artificielles, en pourcentages moyens, d'après les auteurs cités.

En ce qui concerne les résultats de la ménopause roentgénienne, à part le chiffre favorable de GÀL, rapporté plus haut, il ne nous reste à signaler que celui de FELDWEG qui indique un pourcentage de 24.87 % sur 205 cas, et celui de notre statistique, soit 4 % sur 50 cas.

Dans certains cas, nous avons pu noter le phénomène inverse, c. à d. la diminution de la constipation, attribuable sans doute à la régression du fibro-myome.

Donc, le pourcentage le plus élevé des cas de constipation a été noté à la suite de l'ablation des ovaires (CANU) soit: 65.6 %, contre le chiffre le plus élevé à la suite de la ménopause roentgénienne (FELDWEG) soit: 24.87 %.

Récapitulation

Nous avons évalué *la fréquence* des troubles, dans les quatre groupes de ménopause artificielle, en prenant les pourcentages moyens des statistiques que nous venons d'exposer; nous donnons ces pourcentages comparés sous forme de *graphique*, pour en faciliter la lecture (voir ci-contre).

Nous avons laissé de côté pour l'instant, la question de l'hypertension artérielle, en raison de trop grandes discordances dans les résultats publiés jusqu'ici.

Conclusions

Les troubles de déficience ovarienne sont, d'une façon générale, moins fréquents, et en partie moins intenses, dans la ménopause roentgénienne que dans les ménopauses chirurgicales (notamment quand il s'agit de l'ablation des ovaires, avec ou sans hystérectomie). Cela ressort nettement de l'ensemble des données statistiques que nous rapportons.

Peut-on s'appuyer sur cette différence, pour admettre l'hypothèse de la conservation de la glande endocrine de l'ovaire, en cas de ménopause roentgénienne?

Les constatations faites jusqu'ici ne nous paraissent pas apporter de preuves suffisantes pour pouvoir répondre affirmativement à cette question.[1]

Le fait est que les rayons Roentgen, tout en détruisant les éléments parenchymateux des ovaires, les laissent sur place et permettent à l'organisme de les résorber. Il doit se produire de la sorte une *auto-opothérapie ovarienne*, et il est permis de penser que cette auto-thérapie suffise à atténuer la majorité des troubles de la ménopause, et même à empêcher leur apparition.

[1] Sans vouloir entrer ici dans des détails, nous ferons remarquer que les troubles de la ménopause roentgénienne ne varient pas sensiblement avec les diverses techniques employées.

Remarquons à ce propos que la ménopause par hystérectomie semble présenter une certaine analogie avec la ménopause roentgénienne.[1] Les ovaires, il est vrai, finissent par dégénérer, à la suite de l'hystérectomie, ainsi que le prouve la plupart des recherches cliniques et expérimentales, mais ils ne sont pas brusquement supprimés de l'économie, au moment de l'opération, comme en cas d'ovariectomie. Cela expliquerait pourquoi les troubles de ménopause après hystérectomie n'atteignent pas la même fréquence et la même intensité qu'après l'ablation des ovaires, et se rapprochent, au contraire, des résultats de la roentgenthérapie.

RÉSUMÉ

Les auteurs font une étude statistique comparative des troubles de la ménopause artificielle, selon que celle-ci est provoquée par l'ablation des ovaires, avec ou sans hystérectomie, par l'hystérectomie seule ou par la roentgenthérapie.

Leurs observations personnelles sont basées sur 50 cas traités par la roentgenthérapie.

Leur étude porte plus spécialement sur les troubles suivants:

Bouffées de chaleur, hypertension artérielle, adipose, céphalées, asthénie neuro-musculaire, troubles psychiques, diminution de la mémoire, troubles du sens génital, accidents cutanés, constipation.

Les auteurs ne portent pas de jugement pour l'instant sur la question de l'hypertension artérielle de la ménopause, malgré les constatations favorables faites dans leurs cas; car les résultats publiés jusqu'ici sont trop discordants. Mais pour les autres troubles, ils arrivent aux conclusions suivantes:

D'une façon générale, les troubles consécutifs à la ménopause sont moins fréquents et moins intenses après la ménopause roentgénienne qu'après les ménopauses chirurgicales; toutefois les différences sont beaucoup moins prononcées entre l'hystérectomie seule et la roentgenthérapie.

Les auteurs pensent que, dans le cas de la roentgenthérapie, les tissus ovariens détruits restent sur place et sont résorbés par l'organisme, et qu'il se produit ainsi une sorte d'auto-opothérapie ovarienne, suffisante, dans bien des cas, à atténuer la majorité des troubles de la ménopause, ou même à empêcher leur apparition.

SUMMARY

The authors compare the statistics of troubles of artificial menopause, when this is due to extirpation of the ovaries accompanied or not, by hysterectomy alone, or by roentgentherapy.

Their personal observations are based upon 50 cases treated by roentgentherapy.

Their work concerns especially the following troubles:

[1] Nous disons *une certaine analogie:* car, en cas d'hystérectomie nous devons compter, en plus de la possibilité du trauma psychique qui entre en ligne de compte dans chaque type de ménopause chirurgicale, avec les modifications anatomiques consécutives à l'intervention, et peut-être avec la suppression de la sécrétion interne présumée de l'utérus.

Sensation of heat, changes of blood pressure, adiposity, headache, neuromuscular weakness, psychic troubles, diminished memory, changes of the genital sense, skin troubles, constipation.

The authors do not conclude yet concerning arterial hypertension of the menopause, in spite of the favorable results obtained in their cases; for the results published until now are too discordant. Concerning the other troubles, their conclusions are the following:

In general, troubles following the menopause are less frequent and less marked after roentgen menopause than after surgical menopause. There is less difference between hysterectomy alone and roentgentherapy.

The authors think that in the case of roentgentherapy, the destroyed ovarian tissues remain at their place and are absorbed by the organism, producing thus a kind of ovarian auto-opotherapy, sufficient in many cases, to diminish or to prevent the appearance of most troubles of the menopause.

ZUSAMMENFASSUNG

Die Verfasser untersuchen in einer vergleichsstatistischen Studie die nach künstlichem Klimakterium auftretenden Störungen, wobei sie ganz besonders die verschiedenen Kastrationsmethoden in ihrer Auswirkung untereinander vergleichen: Ovariektomie mit und ohne Hysterektomie, isolierte Hysterektomie, und schliesslich Röntgentherapie.

Ihre persönlichen Beobachtungen erstrecken sich auf ein Material von 50 mit Röntgenstrahlen behandelten Fällen.

Die Arbeit der Verfasser berücksichtigt hauptsächlich folgende Ausfallserscheinungen:

Wallungen, Blutdrucksteigerung, Fettsucht, Kopfschmerzen, neuromuskuläre Asthenie, psychische Störungen, Gedächtnisverminderung, Störungen des Geschlechtssinns, Hautveränderungen, Obstipation.

Die Verfasser unterlassen eine Beurteilung der Frage von den klimakterischen Blutdrucksteigerungen trotzdem die in ihren Fällen gemachten Beobachtungen günstig ausfielen, denn die bisher veröffentlichten Resultate sind zu widersprechend. Für die anderen Störungen dagegen, kommen sie zu folgenden Schlussfolgerungen:

Im allgemeinen sind die klimakterischen Störungen nach Röntgentherapie weniger häufig und weniger stark als nach chirurgischem Klimakterium; die Unterschiede sind jedoch viel weniger ausgeprägt bei alleiniger Hysterektomie verglichen mit Röntgentherapie.

Die Verfasser sind der Meinung, dass bei der Röntgenkastration das zerstörte Ovarialgewebe in loco vom Organismus resorbiert wird und so gewissermassen eine ovarielle Autoopotherapie zustande kommt, welche in vielen Fällen genügend ist um die Mehrzahl der klimakterischen Störungen zu lindern oder gar ihr Auftreten zu verhindern.

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MASSIVE (ATELECTATIC) COLLAPSE OF THE LUNG

With a Detailed Discussion of Six Unusual Cases

by

L. R. Sante, M. D., F. A. C. R., F. A. C. P.

Associate Professor of Radiology, St. Louis University School of Medicine, St. Louis, Mo., U. S. A.

(Tabulæ XXIX—XXXII)

Definition and Description. Massive collapse of the lung is a condition in which one or more lobes, previously well aerated, suddenly, without apparent cause, lose their air content and collapse. Sir JOHN ROSE BRADFORD (1) has defined the condition as »an unusual condition in which the lung, without the presence of any gross lesion, such as bronchial obstruction, pleural effusion, etc., interfering with the free entry of air, becomes airless to a greater or less degree». This condition is essentially an atelectasis and should not be confused with collapse of the lung incidental to pneumothorax; the two conditions have nothing in common.

In massive (atelectatic) collapse, the lung does not leave the chest wall. In its collapsed or atelectatic state the lung occupies a smaller space than it did when fully expanded. To compensate for the space lost from collapse of the lung, the chest wall is depressed, the trachea, heart and mediastinal structures are drawn over toward the involved side and the diaphragm is pulled upward. The loss of air content produces consolidation of the lung fully as dense as that of lobar pneumonia.

Radiographic Characteristics. The radiographic picture produced is most striking and quite characteristic. One entire side of the chest shows a dense, homogeneous consolidation. There is a narrowing of the chest cavity on the involved side, the ribs become more oblique and there is a decrease in the intercostal spaces. The trachea, heart and other mediastinal structures are displaced toward the involved side: the tracheal displacement is always evident; the heart may show varying degrees of displacement, ranging from slight displacement to extreme dislocation. The apex beat, normally present in the *left* mammillary line may be displaced to a position in the *right* mammillary line. Where the upper lobe

alone is involved, displacement of the heart may be negligible and deviation of the trachea the only sign. Compensatory emphysema of the uninvolved side may be very marked.

Physical Signs. The physical signs associated with this condition after collapse is well established are likewise very striking. Inspection reveals a flattening and immobilization of one side of the chest. The respiratory function is carried on entirely by the other lung. The percussion note is flat over the entire side and auscultation fails to elicit normal vesicular breathing. The marked displacement of the trachea may give an erroneous impression of pneumonic consolidation.

During the preatelectatic state crepitant and sibilant râles may be heard over the collapsing lung. This was observed in cases IV and VI of our series, but its significance was not understood at the time SCOTT and JOELSON (2) refer to such findings but over both lung fields.

The condition is most frequently encountered as a post-operative complication of abdominal and rectal surgical procedures, especially following appendectomy and herniotomy. Its occurrence is apparently independent of the anaesthetic used, cases being reported following all types of anaesthesia, even spinal anaesthesia, or when local anaesthesia was used. It occurs after fracture of the pelvis, or of the femur, and after injuries apparently trivial to the chest, abdomen or buttocks. The condition may result from post-diphtheritic paralysis of the diaphragm, or from diphtheria, poliomyelitis, or meningitis. It is usually unilateral, occurring most frequently on the same side as the trauma or operation. Numerous cases of contralateral involvement are reported (our Case III is an example). Two instances of bilateral atelectasis have been reported, one by BERGAMINI and SHEPARD (3) following operation for fibroid and death occurred on the operating table. Autopsy revealed total atelectasis of both lungs. The second case was reported by SANTEE (4) as having followed septic abortion and apparently occurred in a physician's office.

Review of the Literature. This condition is by no means a recent observation. It was first reported as a definite clinical entity in 1890 by W. PASTEUR (5) who described it as »massive collapse of the lung». His first observations were on 34 cases of post-diphtheritic paralysis of the diaphragm. In the Bradshaw Lecture (6) in 1908 he discussed the nature of massive collapse of the lung as contrasted with the scattered lobular collapse that may occur when the bronchioles are obstructed by secretions and the alveoli slowly emptied by absorption of their air into the circulating blood. He considered the cause of the collapse as due to paralysis of the diaphragm and other respiratory muscles, and suggested that reflex inhibition of the diaphragm might lead to similar results.

In 1914, he (7) again called attention to the condition as a post-

operative complication of abdominal cases, reporting sixteen such cases which had been encountered in two thousand abdominal operations. He also stated that he had seen a similar condition after injuries to the chest wall. The pathology in every instance was the same.

»Whenever — whether as a result of paralysis or of reflex inhibation of muscular action — the distending force acting on the lungs becomes less than that of the elastic and muscular agencies which tend to cause its contraction, the latter, so to speak, take charge with the result that the affected portion of the lung rapidly empties itself of its contained air.»

The disturbance in the respiratory function may be due either to paralysis or reflex inhibition from inflammation or pain.

In the same year, ELLIOTT and DINGLEY (8) reported eleven cases all of which had followed abdominal operations. After a complete analysis of their cases, these writers came to the conclusion that immobilization of the diaphragm and shallow respiratory movements allowed the bronchioles to become blocked with secretions, the circulating blood absorbing the residual alveolar air. They observed large amounts of mucopurulent sputum in all of their cases and concluded that atelectasis was a direct result of occlusion of the bronchi by this means.

HOLMES (9) cites four cases and refers to twelve others found by BRADSHAW among 3,559 patients operated upon at the Middlesex Hospital from 1906 to 1910. These cases all developed among post-operative patients, especially after appendectomy and herniotomy.

CRYMBLE (10) reported fifteen cases after gunshot wounds did not vary essentially from those resulting from abdominal operation.

In 1918 to 1920 Sir JOHN ROSE BRADFORD (1) made a complete study of the subject in connection with war wounds. He failed to find any postmortem evidence of bronchial obstruction, pleural effusion or other lesion which might interfere with the aeration of the lung, and concluded that, while it seemed incredible, the evidence would seem to indicate that a reflex spasm of the bronchioles was the cause of the obstruction. The condition was observed by him following injuries of the chest and trunk, after fractures of the pelvis or femur, and after gunshot wounds of the chest. He cites an instance where an apparently trivial wound of one side of the chest was followed by a collapse of the opposite lung, no anaesthetic having been administered, and no operative interference undertaken. Some injury, however, seemed to be present in all cases.

BRISCOE (11) in 1920 analyzed the possible causes of massive collapse and concluded that it was produced »by the onset of inflammation affecting the muscles of the crus of the diaphragm, situated behind the peritoneum; one-half of the diaphragm and its synergistic and antagonistic muscles are out of action owing to inflammation of the muscle or the pleural membrane covering it».

In 1921 SCRIMGER (12) reported seven post-operative cases among 540 abdominal and rectal operations; six followed herniotomy or appendectomy, one followed hemorrhoidectomy.

In 1922 ELWYN and GIRSDANSKY (13) reported an instance of massive collapse after a laparotomy for penetrating stab wound of the abdomen. In the same year, HIRSCHBOECK (14) reported three cases following abdominal operations which showed no variation from those already described in the literature. CUTLER and HUNT (15) in this year also made a very painstaking study of post-operative pulmonary complications. They mention massive collapse as a possibility but assert that it never has been observed in their experience.

REGAN (16) in 1924 reported a case of collapse of the lung following poliomyelitis. In the same year RITVO (17) reported an additional case and presented a very complete study of the subject, with special emphasis on the radiological manifestations of the disease.

Early in 1925 SCOTT (18) published a complete study of all reported cases. Aside from war wounds (BARLING and MORRISON (19) & (20)) he found 64 cases in the literature, only 36 of which were given in sufficient detail for analysis. To these he added four cases of his own observation, all occurring as post-operative manifestations, showing little variation from those previously described. Toward the end of the same year, CHEVALIER G. JACKSON and WALTER E. LEE (21) showed the relationship between lobar atelectasis and obstruction of the bronchi by foreign bodies or secretion.

In 1926 and 1927 the writer (22) reported five cases each presenting some unusual feature. These will be discussed in greater detail later on in this communication. During 1926 additional cases were reported and the subject discussed from various angles by MASON (23), JUNGHAGEN (24), TROUT and HOYTER (25), EADES (26), and BEALS (27). None of these reports, however, served to throw any light on the etiology.

In 1927 BERGAMINI and SHEPARD (3) reported two cases of bilateral atelectasis which resulted in death. Both came on immediately following operation. One was in a woman who was operated for fibroid. She was in good condition before operation but died on the table, autopsy revealing bilateral atelectasis. The second case followed septic abortion. SANTEE (4) reported a similar case.

SCOTT and JOELSON (2) call attention to the influence of posture on the development of massive collapse and cite a very remarkable case in which on two separate occasions the same individual was operated for kidney stones on either side and suffered massive atelectatic collapse of the *opposite lung on each occasion*. They cite numerous other instances where the position of the patient may have been a definite factor in producing the collapse. CHURCHILL and HOLMES (28) and (29) stress

the theory that »collapse is the result of a combination of obstruction of the bronchioles by inflammatory oedema and secretion and reflex immobility of the diaphragm». CHURCHILL asserts that bronchial obstruction is essential to collapse. HEARN and CLERF (30) demonstrate progressive reinflation after repeated bronchoscopic examination and removal of mucus. Additional reports and discussions in 1927 were made by BENEDETTI (31), D. Y. and J. P. KEITH and BELL (32), SOLEM (33), MASTICS, SPITTLER and MCNAMEE (34), ASPER (35), CAMPBELL (36), DIEZ (37), GARRETT (38), MCKINNEY and PORTER (39), KLETZ (40), BRENNEMANN (41), LEOPOLD (42), F. J. SMITH and DAVIDSON (43), PEPPARD (44) and others.

Two General Types of Involvement

From a consideration of the reported cases it will be found that the disease manifests itself in two more or less well-defined forms: one in which the symptoms of respiratory distress predominate, the other in which any respiratory symptoms are entirely overshadowed by other manifestations. In the first type, the onset is usually quite sudden, within one to four days after operation or trauma, with pain in the chest, dyspnoea and rapid pulse. The patient may even become cyanotic. The temperature, usually normal at first, later acquires an irregular character, ranging from 98.6° to as high as 102.5° or 103° F, within a few hours. After a few days a cough develops and there may be a small amount of mucopurulent sputum. The degree of the fever seems to run parallel to the quantity of expectoration and to increase as it assumes purulent characteristics. The sputum is never bloody or of the »prune juice» type. The white blood count is somewhat higher than normal, advancing steadily, and with the appearance of mucopurulent sputum reaches as high as 20,000 per cmm.

In the second type the pulmonary symptoms, as has been said, are entirely secondary to other symptoms — either an exciting trauma or operation, or some pain in a distant part. There is no pain in the chest and little if any embarrassment of the respiration or change in the pulse rate. Although the cough, fever and leucocytosis may subsequently develop, they are usually attributed to the inciting condition. In such cases the chest condition is usually discovered incidentally during physical or radiographic examination.

Regardless of the type of involvement, the duration is usually from one to three weeks. The condition may terminate rapidly by sudden reinflation and reestablishment of the respiratory function or may require several weeks for complete recovery. When sudden reinflation occurs, the reexpansion of the lung can be detected immediately in the

radiograph, the lung field taking on a normal appearance in a few minutes. The slower method shows an irregular reestablishment of the respiratory function by successive aeration of small patches of lung tissue, complete reinflation requiring several days or weeks.

Pathology

Very few autopsies are recorded from which the true character of the pathology involved in the condition can be determined. PASTEUR (5) recorded eight autopsies in which massive collapse of the lung followed diphtheritic paralysis of the diaphragm, in five of which massive atelectasis was demonstrated. He was unable to find any bronchial obstruction which could account for the condition in any instance. In every case he noted a thick fibrinous exudate over the diaphragmatic surface and pleura. BRADFORD (1), BRISCOE (11), BERGAMINI and SHEPARD (3), SANTEE (4) and others have made similar autopsy observations in cases which followed war wounds and operations.

BRADFORD failed to reveal any evidence of »gross lesion such as bronchial obstruction, pleural effusion etc., interfering with the free entry of air . . .».

ELLIOTT and DINGLEY (8) emphasize the presence of a large amount of mucopurulent secretion in the bronchioles.

BERGAMINI and SHEPARD failed to find any evidence of obstruction. »Gross examination of the collapsed lung gave no evidence of bronchial obstruction. There were no gross particles in the bronchi. Neither was there any of the mucopurulent secretion so strongly emphasized by ELLIOTT and DINGLEY and by CHURCHILL as a probable etiological factor. Uniform dilatation and engorgement of the capillaries, arterioles and venules strongly suggest probable vasomotor disturbance.»

SANTEE records similar findings.

BRISCOE found a thick fibrinous exudate over the pleura and diaphragm similar to the findings recorded by PASTEUR.

Microscopic section of specimens in all instances showed extensive atelectasis, but in each instance a few rounded globules of air remained.

Case I of our series was autopsied disclosing the same general condition. A similar exudate covered the pleura and microscopic examination of the lung tissue showed atelectasis without cellular reaction. On opening the pleural cavity the chest wall resumed its normal contour and the lung did not fill the chest cavity.

Six Cases Observed by the Writer

I should like at this point to describe six cases which have come under my own observation, not only on account of the rarity of the condition, but also be-

cause each case has certain features which are somewhat unusual. Of these the first five have been previously reported (22).

Case I occurred on the service of Dr. W. T. COUGHLIN, to whom I am indebted for this report.

E. R. K., white, male, aged 5 years, entered the hospital the evening of Feb. 10, 1926 with a history of having been suddenly seized with cramping pains in the abdomen. Nothing in the past history was significant and the only complaint was of intense cramping pains in the abdomen and leg, which had come on a few hours before entrance.

Physical examination revealed pain on pressure over the right lower quadrant of the abdomen and a definite rigidity of the abdominal muscles. Nothing pathologic was found elsewhere. There was »no pain in the chest, no dyspnoea, no symptoms referable to respiratory or cardiac system». The patient was operated on the next morning, Feb. 11, 1926, under ether anaesthesia and the appendix removed through a right rectus incision. The appendix was large and swollen, the end was bulbous and ruptured during removal, allowing a small amount of pus to escape into the peritoneal cavity. A definite localized peritonitis was already present, and a small amount of pus was present in the peritoneal cavity.

The wound was drained and dressed, the patient put to bed, and after a few days rallied and was doing nicely. Drainage became less, drain was removed and the wound granulated nicely; on March 4, patient was permitted to go home.

March 12, 1926, he was readmitted on account of high fever, 103.5° to 104° F. The respiratory rate was high, but there was nothing which would indicate pathology in the chest. Examination of the wound revealed some pus and a grayish-white membrane. Microscopic examination of the pus disclosed diplococci, and blood culture showed streptococci. March 14, an X-ray examination was made of the chest, not because of any definite physical signs or symptoms but to determine any possibility of an associated septic pneumonia.

Some increase in peribronchial markings was noted, but there was no consolidation of the lung or other indication of pathology. The trachea showed slight deviation to the right, but this was not considered, at the time, of any pathologic significance. A second examination was made within thirty-six hours after the first (Fig. 2, Tab. XXIX), which revealed dense consolidation confined to the right upper lobe, with retraction of the trachea toward the involved side. The chest wall showed very little narrowing at that time, and the heart was not definitely displaced. The patient continued to grow worse, running a typical septic course and the pulmonary collapse spread to involve the right and left lower lobes. Nothing, other than pronounced cyanosis, was present, which could be attributed to the lung involvement. The patient died March 19, 1926.

Extracts from autopsy report by the Pathology Department of St. Louis University:

»The lungs do not fill the pleural cavity when the chest is opened. There is a very marked fibrinous exudate throughout both pleural cavities.

The right lung shows almost complete atelectasis of the upper and lower lobes. The middle lobe is relatively increased in size and is rather edematous. The left lung shows atelectasis of the lower lobe and an apparently normal upper lobe. On section, there is no gross evidence of infection, but the lungs exude quantities of blood and fluid.

Microscopic sections of the lungs show complete atelectasis without cellular reaction; other sections show normal expansion of alveoli, with oedema and bronchitis and early broncho-pneumonia.»

The points of special significance to be considered in this case, are: 1) The

fact that massive collapse of the right upper lobe did not occur until thirty-three days after the operation and was probably incident to the general septic condition rather than to the operative procedure. 2) The slight deviation of the trachea *before* consolidation had occurred. 3) The fact that at autopsy, on opening the chest cavity, the lungs did not completely fill the cavity, and the chest wall assumed its normal position.

Case II occurred in the service of Dr. J. G. PROBSTEIN, to whom I am indebted for this report.

O. B., white, male, aged 16 years, entered the hospital May 9, 1926 with a history of having suddenly developed an aching pain in the region of the left hip two days previously, which had become steadily worse until he was unable to stand. The pain came on shortly after an afternoon spent in high jumping, an activity to which he was accustomed. The patient stated that he was sure no injury had resulted; he did not fall or strike his body in any way. Shortly after the pain developed in the hip, the patient vomited, and, on entrance, could not stand, owing to the pain in the hip. Temperature 101.6° F, pulse 80, respiration 24, w. b. c., 11,200 on entrance.

Nothing in the past history was found which could have any bearing on the case. With the exception of a small abscess in the left popliteal space some two years before, there was nothing unusual in the past history.

Physical examination revealed a marked tenderness on pressure over the upper half of the left buttock, »probably, though not necessarily, involving the innominate bone». It was thought at the time that this might be due to a beginning osteomyelitis, since the white blood count was increasing. X-ray examination however, was negative. White blood count: on admission, May 9, 11,200; May 10, 14,800; May 11, 15,200; May 14, 18,000.

May 12, there was some evidence of physical findings in the chest and on May 14, a radiograph (Fig. 3, Tab. XXIX) revealed complete collapse of the entire right lung, with retraction of the heart toward the involved side; narrowing of the chest on this side and elevation of the diaphragm. Fluoroscopic examination showed an immobilization of the diaphragm on the involved side, and a rather hyperactivity of the other.

Fluoroscopic examination three days later showed little, if any, change in the condition. The patient could not sit up on account of pain in the hip, so he was rolled over on the sound side and fluoroscopid in this way. On rolling the patient upon his back for radiographic examination, he complained of sudden pain in the chest, his respiration became shallow and pulse rapid. A cold sweat broke out all over his body. A radiograph was made and it was found that within the few minutes' time elapsing between the fluoroscopic and the radiographic examinations, the entire lower portion of the lung had again expanded and showed apparently normal aeration. The upper portion was still collapsed however (Fig. 4, Tab. XXIX).

The hip condition continued to improve without treatment after this, and on discharge there was little, if any, pain. The lung continued to expand gradually. Examination of the patient three months later (Fig. 5, Tab. XXX) revealed normal expansion and free diaphragmatic movement. The hilus shadows were still pronounced and several rounded structures suggestive of enlarged hilus glands were present. The temperature ranged from 98.6° to 102°, usually not exceeding 100.6° F. It was irregular and was normal at some time every day. There was no fever at all after the third day.

The significant points in this case are: 1) the fact that the condition came on without any wound whatever and without any definite injury; 2) the sudden-

ness with which expansion was reestablished in a large part of the lung merely by rolling the patient upon his side.

Case III occurred in the service of Dr. JOHN STEWART, to whom I am indebted for this report.

JO'C., white, male, aged 30. Entered the hospital Sept. 15, 1926 for kidney fixation operation. His previous symptoms had been entirely referable to the urinary tract. Examination of the chest previous to operation failed to show any evidence of pathology. Kidney fixation operation was done Sept. 18, under ethylene-ether anaesthesia. The post-operative healing of the wound was uneventful, but there was some elevation of the temperature. On Sept. 25, the seventh post-operative day, the patient developed a slight cough and his temperature rose as high as 102.8° F. The white blood count at this time was 8,250. Physical examination made by Dr. RALPH KINSELLA, was reported as follows: ». . . dullness in the left upper lobe, anterior and posterior. Whispered voice sound not increased over this area. Sonorous râles and whistles over the right chest, anterior and posterior. Pericardial friction rub heard over one inch lateral to the nipple on the left. Dullness over the left, marked involvement of the entire side.» Massive collapse was suspected and this diagnosis was made prior to X-ray examination.

On the 26th of Sept. the first radiographic examination was made (Fig. 6, Tab. XXX) showing a dense homogeneous consolidation involving the entire left side, with narrowing of the chest wall, elevation of the diaphragm and retraction of the heart and trachea toward the involved side. Bronchoscopic examination failed to reveal any foreign body, but a quantity of mucus was evacuated. Following bronchoscopy (performed by Dr. I. B. KELLY) the patient was rolled over on the uninvolved side and instructed to cough. Immediately the lung began to inflate under direct fluoroscopic vision (Fig. 7, Tab. XXX), starting first at the costo-phrenic angle and periphery of the lower lobe, which was soon followed by some inflation of the medial border also. Simultaneous with reinflation of the lung, the interspaces became wider and the mediastinal structures assumed a more natural position. After this, the condition progressed rapidly toward reinflation, and, with the exception of a small area of atelectasis in the upper lobe, the entire lung was reinflated within seven days (Fig. 8, Tab. XXX). However, even at this time, fluorsscopic examination of the diaphragm revealed complete immobilization in a high position. The diaphragm was not paralyzed, there was no paradoxial action — it was simply immobilized in a high position. The patient went on to an uneventful recovery.

The significant points in this case are: 1) The occurrence of the lung collapse on the opposite side from that upon which operation was performed, 2) the bronchoscopic examination which failed to reveal any definite obstruction, 3) the rapid inflation under direct fluoroscopic vision after rolling the patient back and forth on the unaffected side and causing him to cough, and 4) the observation that immobilization of the diaphragm persisted long after inflation was completely reestablished.

Case IV occurred in the service of Dr. TAINTER at the St. Louis City Hospital, to whom I am indebted for this report.

J. S., a white man, aged 23, entered the hospiral, Jan. 6, 1927, following gunshot wounds of the abdomen, pelvis and left wrist. Under ether anesthesia the abdomen was explored for possible perforation on the 7th of Jan., but none was found, and the abdomen was closed in the usual manner with drainage of subcutaneous tissue. A chest film taken before operation did not show any evidence of a pathologic condition. Convalescence was uneventful until the fourth day after operation, when the patient complained of shortness of breath

and pain in the right half of the chest and in the region of the umbilicus. Physical examination at this time disclosed dense consolidation of the right lung; and since the temperature was somewhat elevated, 102° F, and the respiratory rate somewhat increased, 40 a minute, a diagnosis of post-operative pneumonia was made. Roentgenographic examination, however, revealed the characteristic condition of massive collapse, dense consolidation of the entire right lung, narrowing of the right side of the chest, approximation of the interspaces and elevation of the diaphragm. The heart and mediastinal structures were displaced to the right side to such a degree that the heart was not visible to the left of the spine. The apex beat was best heard in the right anterior axillary line (Fig. 9, Tab. XXX).

Fluoroscopic examination, Jan. 19, still revealed the massive consolidation without apparent change. While under fluoroscopic examination, the patient was instructed to roll back and forth on the uninvolved side and to cough. In this instance, as in all others previously reported, the collapsed lung expanded during fluoroscopic examination. Within a very few minutes, almost complete reinflation was established (Fig. 10, Tab. XXXI). The diaphragm on the right side was immobilized in a high position. During the process of reinflation, a small amount of mucus was coughed up and expectorated into a handkerchief. The amount did not exceed 5 cc, and could hardly have explained the obstruction or the excessive density of the lung in its collapsed state. It was globular and gray, and possessed the same gum-drop appearance described by JACKSON and his coworkers.

Reinflation proceeded without further difficulty. Although there was a tendency to further collapse on remaining quietly in bed, rolling the patient quickly reestablished inflation (Fig. 11, Tab. XXXI). This case is reported since it presents a complete radiographic record with roentgenograms before collapse, during the active stage of the disease, and reinflation; and since it further confirms the efficacy of the treatment which I have previously recommended.

The points of significance in this case are: 1) a chest radiograph showing normal lung structure was available before the collapse, 2) reinflation was again observed in three to four minutes under direct fluoroscopic vision, after rolling the patient upon his uninvolved side, 3) the cardiac shadow previously drawn over to a position far beyond the midline was seen gradually to resume its normal position, 4) the sequence of events incident to reexpansion was noted: aeration began at the periphery in the lower portion of the chest; the trachea was the first structure to resume its normal position in the midline, its shadow becoming narrower as it regained its proper position; with continuing reexpansion the cardiac shadow began to resume its normal position and the ribs to lose their abnormal, oblique position. Even after complete reinflation the diaphragm remained immobile in a high position, 5) the tendency to recollapse was noted if the patient was permitted to remain on the involved side for any great length of time.

Case V occurred on the service of Dr. WILLIAM G. HERRMANN who made the examination and to whom I am indebted for this report.

E. F., a thin, somewhat emaciated, white, male patient, aged 19. On admission, Dec. 22, 1926, he showed marked dyspnoea and cyanosis and had to sit up in bed to »get his breath». Nothing in the history was of significance, other than periodic attacks of asthma for the past several years. Just prior to his admission to the hospital, while suffering from an asthmatic attack, he had experienced a sharp pain in the chest.

Extract from physical examination submitted by Dr. HERRMANN:

»... physical examination at this time revealed immobility of the entire left

chest. This was very marked und quite striking on inspection of the chest. Tactile fremitus absent throughout the entire left chest with exception of a small area posteriorly about the level of the spine of the scapula. Percussion showed a hyper-resonant note amd a tendency to tympany over the first, second and third interspaces, anteriorly, and over the area extending from apex to about the level of the spine of the scapula posteriorly. Below this, there is absolute dullness. Auscultation revealed absence of breath sounds throughout the left chest. Right chest negative, with the exception of a few mucous râles diffusely distributed. The apex beat of the heart was in the fifth interspace in the anterior axillary line, regular in rate and rhythm.»

The temperature at first ranged as high as 103.5° F. but later fell gradually and became normal on Dec. 24.

Radiographic examination immediately after administration revealed massive collapse of the left lung with associated pneumothorax. Since the existing pneumothorax was sufficient partly to equalize the pressure caused by the collapse of the lung (Fig. 12, Tab. XXXI), no narrowing of the side of the chest, displacement of the mediastinum or elevation of the diaphragm were produced; there was however, a slight tracheal displacement toward the involved side. Two days later, Dec. 24, the patient was brought to the fluoroscopic room and rolled over on the unaffected side (Fig. 13, Tab. XXXI). The lower lobe cleared, and a small, rounded area of aeration was noted in the consolidated lobe. The upper lobe cleared partially, also, but the pneumothorax was still quite evident (Fig. 14, Tab. XXXI). Three days later examination revealed almost complete reinflation of the lung, with only a very slight pneumothorax remaining (Fig. 15, Tab. XXXII.) The patient went on to an uneventful recovery and left the hospital Jan. 5, 1927.

The unusual aspects of this case are: 1) the association of pneumothorax with atelectasis, 2) the failure of the secondary phenomena (displacement of mediastinum and narrowing of the chest) to develop owing to the pneumothorax, 3) the reinflation of the atelectatic lung after rolling the patient upon the uninvolved side, even when the atelectasis was complicated by pneumothorax, and 4) the inciting cause due to asthma.

Case VI is here presented for the first time and is unique in that it represents a complete radiographic record of this condition from the time of injury, throughout the developing collapse, to complete subsequent inflation. Case VI occurred on the service of Dr. Cole, to whom I am indebted for this report.

A. G., white, male, aged 20, was admitted to the hospital at 4.45 a. m., on Jan. 18, 1928, shortly after the automobile which he was driving had been struck by a railroad train. Physical examination disclosed multiple abrasions, contusions and lacerations of the scalp, body and extremities. X-ray examination revealed the 12th rib on the right side completely pulled from its spinal attachment and displaced about an inch to the side. There was no evidence of other injury to the skull, spine or chest, no evidence of a hollow viscus (no free air in the abdomen) and *the diaphragms were in normal position.* Shortly after admission, the patient passed blood in the urine. On admission, temperature was 98.4° F, pulse rate 112, respirations 24 per minute, blood pressure 115/55, w. b. c., 7,000.

On the afternoon of the same day (Jan. 18) radiographic examination of the chest was made (Fig. 16, Tab. XXXII). In the few hours intervening between these examinations, the right diaphragm had become elevated, and was immobile; there was also an irregular accentuation of the lung markings throughout the right chest. Physical examination made the morning of Jan. 19 (scarcely more than 24 hours after the injury) revealed a flat percussion note over the entire right chest.

Radiographic examination on January 20 showed a typical picture of massive collapse of the lung (Fig. 17, Tab. XXXII). During the night, the pulse rate reached 140 p. m., and the respirations were as frequent as 40 per minute. Within a few hours these dropped to a pulse rate of 88 and respiration rate of 20. Further radiographic examination, made January 23, revealed almost complete reexpansion with only a very small atelectatic area remaining in the lung (Fig. 18, Tab. XXXII). No therapeutic measures were attempted, other than the ordinary rolling about in bed.

The points of interest in this case were: 1) the complete record afforded by examination within a few hours after injury, showing the diaphragmatic position normal, 2) the radiographic record in the films taken within ten hours after injury, showing the beginning of collapse, 3) the evidence of complete collapse within 24 hours after injury, and 4) the spontaneous reexpansion.

Etiological Theories

From a consideration of all reported cases, four hypotheses have been advanced as possible explanations of the cause of this condition:

1. The Paralytic Theory, that the collapse of the lung is due to pressure exerted by the collapsed chest wall and elevated diaphragm, resulting in turn from paralysis of these structures. That the resulting sluggishness of respiration permitted collection of secretion in bronchioles which caused this obstruction, and that the air remaining in the alveoli of the obstructed area was absorbed by the circulating blood. In other words, the pressure of the chest wall and diaphragm is considered primary, the lung collapse, secondary. (Supported especially by PASTEUR.)

2. The Diaphragmatic Infection Theory, »that due to the onset of inflammation affecting the muscles of the crus situated behind the peritoneum, one-half of the diaphragm and its synergistic and antagonistic muscles are out of action, owing to inflammation of the muscle or the pleural membrane covering it». This results in a disturbance of the respiratory function which in turn leads to collapse. (The view held by BRISCOE.)

3. The Spasmodic Reflex Theory, that some spasmodic reflex of the bronchioles causes them to become occluded, permitting the subsequent absorption of air in the segment of the lung involved. (Explanation advanced by Sir JOHN R. BRADFORD.)

4. The Simple Obstruction Theory, that obstruction of the bronchioles by secretions is the entire explanation such a condition being all that is necessary for the development of the other features of the condition, and any disturbance of the respiratory function, purely incidental. (Observations of JACKSON and LEE.)

5. The Angioneurotic Theory, that the rapidity of development of the condition in certain instances and the dilatation of the capillaries and engorgement of the blood vessels lend credence to this theory, ac-

cording to BERGAMINI and SHEPARD. Both GWYN (45) and SCOTT also mention this possibility.

6. The Posture Theory, that the position assumed by the patient during post-operative convalescence may favor the production of this condition. This view is held by SCOTT and JOELSON.

7. The Theory of Combined Obstruction and Impaired Respiration, that »Collapse is the result of a combination of obstruction of the bronchioles by inflammatory oedema and secretion, and reflex immobility of the diaphragm», the view held by CHURCHILL, and also mentioned in substance by ELLIOTT and DINGLEY.

Clinical Observations, Pathological Findings, and Experimental Evidence, Tending to Support or Contradict These Theories

The original contention of PASTEUR that this atelectatic condition was due to the pressure of the depressed chest wall which resulted from paralysis of the diaphragm and other respiratory muscles, is not borne out by the facts. In patients suffering from tuberculosis, in whom one of the phrenic nerves has been sectioned as a therapeutic measure, this condition has never resulted; consequently some other factor must have been a contributing cause in PASTEUR's original cases. ELLIOTT and DINGLEY performed experiments on rabbits sectioning the phrenic nerve and other nerves influencing respiration and never witnessed this phenomenon. BRISCOE likewise performed section of the phrenic nerve with similar results, while CORYLLOS and BIRNBAUM (46) failed to produce the condition after phrenicotomy.

It will be recalled that at the autopsy of our first patient, on opening the chest cavity, the chest wall assumed its normal contour and the lungs failed to fill the entire cavity. This would indicate that the depression of the chest wall was secondary to lung collapse and not the primary factor in producing this lung condition. In Case V of our series also, where atelectasis was associated with pneumothorax the chest wall was never retracted. ELKIN (47) measured the intrathoracic pressure in patients suffering with massive collapse and found the negative intrapleural pressure increased. Normally the intrapleural pressure varies from —9 mm Hg on inspiration to —2 mm Hg on expiration (48). In the three cases cited by ELKIN, the negative pressure was more pronounced in both chest cavities. The greatest variation noted was —12 mm Hg on inspiration and —15 mm Hg on expiration on the involved side; —6 mm Hg on inspiration and —8 mm Hg on expiration on the uninvolved side. In the roentgenographic observations made in Cases I and VI, there was retraction of the trachea before definite indications of collapse of the lung or sinking in of the chest wall. In Case V, where spontaneous

pneumothorax occurred in association with massive atelectatic collapse the chest wall did not become sunken, and there was little if any displacement of the walls of the pleural cavity. We must conclude, therefore, that massive collapse of the lungs does not occur solely as a result of paralysis of the diaphragm or other respiratory muscles, nor from pressure of the chest wall.

BRISCOE's supposition that collapse is produced by a lack of synchronism between the diaphragmatic movements brought about by retroperitoneal infection of the crus of one or other diaphragm, cannot be upheld because at best it would explain abdominal cases only, and post-operative cases in which there had been infection. Many cases have been cited where the condition came on so quickly after trauma (gunshot wound) that infection should be impossible. The cases reported by BERGAMINI and SHEPARD and by SANTEE in which collapse occurred during operation, would rule out infection as an indispensable cause. In Cases II and VI of our series, also, collapse followed so soon after injury as to make infection impossible. BRISCOE's basis for this assumption of infection as a cause lies in the detection at autopsy of a thick fibrinous exudate over the pleura and diaphragm. A similar exudate was noted by PASTEUR in his autopsies of post-diphtheritic patients and in the autopsy which was performed in Case I of our series this finding was also confirmed. In all of these cases, however, a previous infection had been established. No evidence of such an exudate was present in any of our cases in which sudden complete reinflation occurred. Complete, rapid reinflation was noted in Cases II, III, IV and VI and in none of these was there evidence of such an exudate. Reinflation was so rapid that there would have been no time for the absorption of an exudate had it been present. The autopsy reports of BERGAMINI and SHEPARD and of SANTEE on their cases do not mention this finding.

We must conclude, therefore, that surely, in many cases, infection of the diaphragm plays no part in the production of collapse and that in such cases where a thick, pleural exudate does form over the diaphragm and pleura, it is rather the result of inactivity of the diaphragm than the cause of any interference with its motion. In every case reported in the literature, the appearance of an exudate is in association with an established infection.

The theory advanced by BRADFORD, that collapse of the lung is brought about by a reflex stimulation of the constrictor nerve fibers to the muscles of the bronchioles, remains still to be considered. Ample experimental evidence is found of the existence of constrictor nerves and muscle fibers on the bronchioles sufficiently strong to produce complete and prolonged constriction (LONGET (49); AUER and LEWIS (50)). That such constriction ever results in atelectasis, however, has never

been proven. The frequency with which the condition follows abdominal operation would lead one to suspect that an insult to the vagus nerve might offer the reflex stimulus for this occurrence. BRISCOE, however, performed numerous experiments in which in an attempt to produce this reflex spasm he irritated the peritoneum, stimulated the vagus, etc., all without results. There is, therefore, no direct evidence in support of the view that reflex *bronchial spasm* is the cause of the disease, although there is evidence to indicate that *contractions of the bronchial musculature* may be sufficiently powerful to shut off the bronchi.

Bronchial obstruction by secretion alone is the cause suggested by JACKSON and LEE. Long before the discovery of this condition it had been observed that when a foreign body completely occluded a bronchus, the portion of the lung supplied by the obstructed bronchus became atelectatic. MENDELSSOHN (51) in 1845, and in the following year, TRAUBE (52), produced atelectasis by occluding the bronchi with paper wads, shot and acacia. LICHTHEIM (53) in 1879 found that introduction of a stick of laminaria into a bronchus of a rabbit caused atelectasis of its lung segment within a few hours, provided the circulation of the blood was left intact. If the blood vessels to this section of the lung were ligated simultaneously with the obstruction of the bronchus, the air remained in the obstructed lung and atelectasis did not ensue. From this it was concluded that the residual air is rapidly absorbed by the circulation. Similar observations by CHEVALIER JACKSON and his co-workers on foreign-body atelectasis in human beings, has resulted in the same conclusions. Clinicians (GOLDEN (54); CHIZZOLA (55)) have observed that bronchial tumors, when they completely obstruct, produce within a very short time a complete atelectasis of the segment of the lung supplied by the bronchus.

Partial obstruction results in emphysema. Since the diameter of a bronchus is smallest during expiration, obstruction first occurs during this phase of respiration; the air which is drawn in during inspiration cannot subsequently be expired and remains trapped. This phenomenon is the basis of MANGES's (56) diagnostic sign for non-opaque foreign bodies in the lung. Obstructive emphysema is caused by the partial obstruction of a bronchus. The excellent experimental work of CORYLLOS and BIRNBAUM in which a bronchus was obstructed to varying degrees by inflation of balloons introduced through a bronchoscope, serves to confirm these previous observations and to establish beyond a doubt the relationship between complete bronchial obstruction and atelectasis, but leads no closer to a solution of the problem in spontaneous »massive collapse« of the lung, where no evidence of foreign-body obstruction has been found.

Aspiration of secretions alone could hardly explain the condition

since patients, aspirating barium sulphate through broncho-oesophageal fistulae, never develop atelectasis. Likewise, no case is reported after tonsillectomy and there is abundant proof of the extreme degree of aspiration which frequently occurs during this operation. In no recorded autopsy was there any evidence of bronchial obstruction.

That it is possible for thick fluids to result in atelectasis under certain conditions is demonstrated in a case cited by HICKEY, according to a recent personal communication by Dr. PRESTON B. FORESTIER. In HICKEY's patient, massive collapse followed the intratracheal injection of iodized oil used for diagnostic purposes.

The rapidity with which the condition develops in certain instances and the engorgement and dilatation of the capillaries suggests the possibility of vaso-motor disturbance as a contributing etiological factor. GWYN mentions that massive collapse may be due to some such action and SCOTT also is impressed with this possibility. BERGAMINI and SHEPARD state that the »uniform dilatation and engorgement of the capillaries, arterioles and venules strongly suggest a probable vasomotor disturbance». The rapid onset in the cases observed by them »would seem in itself to rule out the theory of obstruction of the bronchi with subsequent absorption of the alveolar air». They suggest some such explanation as angioneurotic oedema.

Posture alone as an etiological factor has been stressed by SCOTT and JOELSON. To cite one notable case on which massive collapse occurred in the contralateral lung in two successive occasions after operation on the two kidneys. BRISCOE had previously suggested position as a possible cause.

The theory of combined obstruction and impaired respiration is the view upheld by CHURCHILL. He asserts that there can be no atelectasis without bronchial obstruction.

When we consider the facts, we have no clinical or experimental evidence for thinking that atelectasis can occur under any other condition than actual mechanical bronchial occlusion. If we assume that there can be no atelectasis without previous bronchial obstruction, then the manner in which the obstruction is brought about alone remains to be considered. What extraneous influences are there which could account for the accumulation of secretion in the larger bronchi? Obviously, the loss of sensation of the trachea and larger bronchi and the consequent interference with the expulsive mechanism which is normally present — cough reflex and ciliary action of the mucous membrane. The cause of such loss of sensation on the part of the trachea and bronchi, then, becomes the chief point of consideration; atelectasis is merely the natural course of events which would be expected under these circum-

stances. Will loss of bronchial sensation explain all instances and circumstances under which the condition is found?

Infectious cases might well be explained in this manner. There is abundant evidence that extreme toxicity can abolish the tracheal reflex. This is seen in instances where individuals toxic from intestinal obstruction or septicaemia, aspirate barium sulphate into the lungs without discomfort. PASTEUR's original cases following diphtheria, cases reported following acute poliomyelitis and Case I of our own series in which the causative factor was probably septicaemia, are examples. The loss of tracheal reflex in operative cases might also be explained either by anaesthesia produced by morphine, or other hypnotics given to quiet the patient, especially if he were permitted to remain in one position for an extended period of time after the operation. Preoperative administration of morphine has been offered as a possible explanation of post-operative abscess of the lung, the analgesia produced preventing the normal expulsion of infected material aspirated during operation. This theory would explain contra-lateral involvement which is occasionally encountered in post-operative atelectasis as seen in Case III of our series. The case referred to by Dr. HICKEY in which massive collapse followed intratracheal injection of iodized oil, might well have been due to the previous injection of cocaine, administration of morphine, or large doses of luminal (which is supposed to counteract the constitutional effect of cocaine) prior to injection and permitting the patient to lie quietly after injection. But toxicity *alone* will not explain all cases in which the phenomenon occurs.

There remain for consideration those cases in which the condition develops after injury (Cases IV and VI of our series), and severe physical strain. It is conceivable that some reflex might result from injury to a remote part of the body. It is not uncommon to see patients so severely injured in one part of the body that the pain from a less severe injury elsewhere is inhibited or overshadowed. During the war it is possible that severe fighting conditions, high nervous tension and extreme exhaustion incidental to battle, might result in the inhibition of the cough reflex. Numerous instances have been encountered during the war and in civil life where abdominal operations were performed without an anaesthetic upon individuals in a profound state of exhaustion. In the strain of competitive athletics could not the great nervous tension and muscular effort (Case II) produce such reflex inhibition from fatigue?

The one constant finding in all instances is immobilization (not paralysis) of the diaphragms. While the collapse itself is often on the opposite side to the operation or injury which precedes the condition, the diaphragmatic immobilization is always on the same side as the collapsed lung. If aspiration of secretions alone were the cause, diaphragmatic

excursion should be resumed after the dislodgment of the obstruction and reinflation of the lung. This is not found to be the case; diaphragmatic immobilization often remains for days or weeks after reinflation. This would seem to indicate that it is inactivated in a defense reflex.

Is it not conceivable that the bronchioles might also be contracted from a similar reflex spasm? Sibilant râles have been detected by others intermittently over the lung during the time preceding collapse, and the same observation has been made by clinicians in our cases before it was known that collapse was developing. May it not be possible, then, that collapse occurs as a result of a combination of all of the factors here noted?

It seems most logical to suppose from the available evidence that massive atelectatic collapse of the lung results from a simultaneous inhibition of the cough reflex by some toxic or reflex stimulus in association with an impairment of the respiratory function, either immobilization of the respiratory muscle from a defense reaction, or paralysis from toxic neuritis. This in turn permits the accumulation of secretions, blocking the bronchi and resulting in atelectasis. Posture favors the development in the most dependent portion. Occurring alone, these factors may not be sufficient to produce the condition; their simultaneous occurrence, however, may be all that is necessary to result in collapse.

Differential Diagnosis

Massive (atelectatic) collapse of the lung must be differentiated from

1) Lobar pneumonia
2) Lung abscess
3) Caseous tuberculous pneumonia
4) Broncho-pneumonia
5) Lung tumor
6) Pleural effusion
7) Chronic interstitial pneumonia

If one bears in mind the clinical history and the typical roentgen picture of massive (atelectatic) collapse, none of these conditions should cause confusion.

Massive (atelectatic) collapse most frequently follows abdominal operations or other injury to the chest, abdomen, or pelvis. The onset is sudden with pain in the chest, fever, dyspnoea, rapid pulse and leucocytosis. There are the physical signs of massive consolidation. At this stage the condition may resemble lobar pneumonia, especially in so far as pneumonia is a frequent post-operative complication. The one distinctive sign is the retraction of the heart and mediastinal structures toward the involved side and sinking in of the chest wall on that side. Lobar pneumonia does not, under any circumstances, produce deviation of the trachea or displacement of the mediastinal structures. If only a single lobe, for instance the upper, is collapsed, the phenomenon of

sinking in of the chest and pulling over of the heart does not occur, since the slight difference in space caused by collapse of a single lobe is compensated for by emphysema of the remaining aerated lung. Even in this instance, however, there will be definite displacement of the trachea which will establish the true nature of the condition. If there is ever doubt about the consolidation being due to lobar pneumonia, lung abscess, caseous tuberculous pneumonia, or any consolidation other than massive collapse, a safe diagnostic procedure is to roll the patient over upon his uninvolved side and cause him to cough. An atelectatic lung will immediately reinflate; consolidation from other cause will not be affected.

After a few days the patient begins to cough, and after a time brings up a varying amount of mucopurulent sputum. At this stage the condition may be mistaken, clinically, for lung abscess or caseous tuberculous pneumonia. Roentgenographically, however, the picture is usually quite distinctive. The formation of secretion, it should be remembered, is one of Nature's methods by which inflation is reestablished, so that at this stage, patches of reinflation may be seen here and there in the atelectatic area, and the picture may become confused. With beginning reinflation of the lung, the first structure to return to normal position is the trachea, so that this might have resumed its position in midline if there is much evidence of reinflation. The narrowing of the interspaces and shallowness of the chest on the affected side would still be present to establish the diagnosis, however. At a somewhat later stage, where the process has been allowed to go on to reinflation by degrees, a stage is reached in which all of the displaced structures again assume their normal positions, and even at this stage there may be considerable irregular atelectatic areas remaining in the lung. It may, just at this time, be impossible to differentiate the condition from broncho-pneumonia. Massive collapse, however, is unilateral, and broncho-pneumonia is usually bilateral.

Tumours of the lung may reach such enormous proportions as to completely fill the chest. Such tumors may continue to grow until they cause a displacement of the heart and mediastinal structures to the opposite side, but they never cause retraction toward the involved side.

Large pleural effusions, likewise, may produce a homogeneous shadow involving one entire side of the chest. In this condition, displacement of the heart and mediastinal structures away from the involved side is the rule. Such displacement may not be present due to fixation of the mediastinum from previous infection, but retraction of the mediastinal structures toward the involved side never results. Occasionally a long-standing pleural effusion may have developed sufficient scar tissue in its surrounding capsule to cause retraction of the trachea, but this is

very rare, and the other elements of the case will usually establish the true nature of the process.

Chronic interstitial pneumonia (chronic diffuse fibrosis of the lung) is the only other massive consolidation which causes retraction of the heart and mediastinal structures *toward* the involved side. This condition is chronic, however, usually following some suppurative process in the chest and the scar tissue which causes the pulling over of the mediastinum likewise exerts its traction on the walls of the bronchi, causing bronchiectatic cavities to form. These can be clearly seen through the consolidation. They never occur in association with massive atelectatic collapse.

Treatment

The fluoroscopic observation of immediate reinflation of the collapsed lung after rolling the patient back and forth on the uninvolved side (affected side uppermost) and causing him to cough, gives a logical basis for this procedure as a therapeutic measure. This simple expedient has resulted in immediate reinflation in every instance which we have observed, and numerous confirmation of the observation have been brought to the writer's attention in this country and abroad. CHEVALIER JACKSON and his co-workers, HEARN and CLERF, have shown that reinflation occurs following bronchoscopic removal of mucus from the bronchi. The process of reinflation may be slow, however, and often is complete only after several bronchoscopic examinations. It would seem advisable, then, in all instances to attempt the simple expedient of rolling the patient on the uninvolved side and causing him to cough before bronchoscopic procedure is instituted. Should this measure fail to produce reexpansion, bronchoscopic examination should be resorted to, in order to detect any mechanical cause for the collapse. Dr. JACKSON and members of his clinic have demonstrated numerous instances of atelectasis from bronchial occlusion by new growths, foreign bodies and anomalies of the bronchi; these conditions represent atelectasis from mechanical obstruction and are in no way similar to the cases here under discussion. Obviously simple change in position would have no effect on the atelectasis produced under such circumstances and bronchoscopic examination would be indicated for the detection of its cause.

SUMMARY

1. Massive atelectatic collapse of the lung is a definite clinical entity. This name should be reserved for cases following out the general clinical course herein mentioned, in which there is collapse of a previously well-aerated lung without mechanical obstruction from foreign body, tumor or anomaly. This name is not

entirely descriptive of the condition and will probably give place in time to a more scientific designation. The term »acute lobar idiopathic atelectasis» has been suggested by Dr. SETH I. HIRSCH.[1] Congenital atelectasis and atelectasis from mechanical cause such as foreign bodies, tumors and anomalies should not be so considered.

2. It is characterized by sudden collapse of one or more lobes in a lung previously well aerated, from some unknown cause.

3. It is most frequently observed after abdominal operations, wounds and other injuries such as fractures of the pelvis or femur, but may follow apparently trivial injuries.

4. Roentgenographically, there is a dense consolidation corresponding to one or more lobes or to an entire lung, homogeneous in character and resembling consolidation from pneumonia. The narrowing of the chest on the involved side, the approximation of the intercostal spaces, the elevation of the diaphragm and the drawing over of the heart and mediastinal structures toward the involved side, make the condition readily recognizable.

5. It seems most logical to suppose that the condition is due to a simultaneous inhibition of the cough reflex by some toxic or reflex stimulus in association with an impairment of the respiratory muscles, either immobilization from a defense reaction or paralysis from toxic neuritis, which permits secretions to accumulate and block the bronchus, and atelectasis results. Neither alone is able to bring about this condition; a simultaneous occurrence is necessary for its development.

6. The treatment is simple and consists in rolling the patient back and forth on the uninvolved side. Ordinarily no other therapeutic agent is necessary. This simple procedure has, in all instances in which we have instituted it (five) proved successful in promptly reestablishing aeration of the lung. JACKSON and his co-workers have reported reinflation after repeated bronchoscopic removal of mucus from the bronchi and bronchoscopy should be resorted to if this simple maneuver fails.

ZUSAMMENFASSUNG

1. Massiver atelektatischer Kollaps der Lunge ist eine wohl definierte klinische Entität. Dieser Name sollte für Fälle vorbehalten werden, die dem allgemeinen klinischen, hier geschilderten Verlauf folgen, in dem Kollaps einer früher gut lufthaltigen Lunge ohne mechanische Obstruktion durch Fremdkörper, Tumor oder Anomalie vorliegt. Die Benennung ist nicht völlig bezeichnend für den Zustand und wird mit der Zeit wahrscheinlich durch einen wissenschaftlicheren Terminus ersetzt werden. Dr. SETH I. HIRSCH[2] hat den Namen »akute lobäre idiopathische Atelektase» vorgeschlagen. Kongenitale Atelektase und Atelektase aus mechanischen Ursachen wie durch Fremdkörper, Tumoren und Anomalien würden dadurch nicht in diesen Krankheitsbegriff einbezogen sein.

2. Der Krankheitszustand ist charakterisiert durch plötzlichen Kollaps eines Lappens oder mehrerer in einer früher gut ventilierten Lunge, aus unbekannter Ursache.

3. Am häufigsten wird er nach abdominalen Operationen, Wunden und anderen Traumen wie Frakturen der Pelvis oder des Femur beobachtet, er kann aber auch im Gefolge anscheinend unbedeutender Schädigungen auftreten.

[1] Personal communication.

[2] Persönliche Mitteilung.

4. Röntgenologisch findet sich entsprechend einem Lappen oder mehrerer oder einer ganzen Lunge eine intensive Verdichtung von homogenem Character, ähnlich der Verdichtung durch eine Pneumonie. Die Verengerung des Brustkorbes an der befallenen Seite, die gegenseitige Annäherung der Interkostalräume, der Zwerchfellhochstand und die Hinüberziehung des Herzens und der Mediastinalgebilde gegen die befallene Seite machen den Zustand leicht erkennbar.

5. Am logischsten erscheint die Annahme, dass der Zustand zurückzuführen sei auf eine Verhinderung des Hustenreflexes durch einen toxischen oder reflektorischen Reiz in Verbindung mit einer Funktionsstörung der Respirationsmuskeln, entweder einer Immobilisierung durch eine Abwehrreaktion oder einer Lähmung durch toxische Neuritis, wodurch es ermöglicht wird, dass die Sekrete sich ansammeln, den Bronchus verstopfen und so zu Atelektase führen. Keiner der beiden Faktoren ist imstande, den Zustand allein zu erzeugen; zu seiner Entwicklung ist ein gleichzeitiges Zusammenwirken beider notwendig.

6. Die Behandlung ist einfach und besteht darin, dass der Patient auf der gesunden Seite hin- und hergerollt wird. Gewöhnlich ist keine andere therapeutische Massnahme erforderlich. Diese einfache Prozedur hat sich in allen (fünf) Fällen, in welchen wir sie angewendet haben, erfolgreich gezeigt, indem sie prompt die Lüftung der Lunge wiederherstellte. JACKSON und seine Mitarbeiter haben über Reinflation nach wiederholter bronchoskopischer Entfernung des Schleimes aus den Bronchi berichtet, und wenn das oben erwähnte einfache Manöver im Stich lässt, sollte Bronchoskopie versucht werden.

RÉSUMÉ

1. Le collapsus massif atélectasique du poumon constitue une entité clinique bien définie. Cette dénomination doit être réservée aux cas présentant l'évolution clinique mentionnée ici et où il s'agit de collapsus d'un poumon antérieurement bien aéré, sans obstruction provenant d'un corps étranger, d'une tumeur ou d'une anomalie. Le nom donné à cette affection n'est pas entièrement descriptif de sa nature et fera probablement place ultérieurement à une dénomination plus scientifique. Le terme »d'atélectasie lobaire idiopathique aiguë» a été proposé par le Dr SETH I. HIRSCH[1]. L'atélectasie congénitale, pas plus que l'atélectasie d'origine mécanique, par suite de corps étranger, de tumeur ou d'anomalie, ne répondent pas à cette dénomination.

2. L'affection est caractérisée par un collapsus subit d'un ou plusieurs lobes d'un poumon antérieurement bien aéré, sans cause connue.

3. Elle s'observe surtout à la suite d'opérations abdominales, blessures ou autres lésions telles que fracture du bassin ou du fémur, mais peut également survenir à la suite d'une lésion insignifiante.

4. Radiographiquement, l'affection présente l'aspect d'une hépatisation dense intéressant un ou plusieurs lobes pulmonaires ou même la totalité du poumon, de caractère homogène et rappelant celle de la pneumonie. Le rétrécissement du thorax du côté involué, le rapprochement du bord costal, l'élévation du diaphragme, le déplacement du coeur et des organes médiastinaux vers le côté malade rendent l'image clinique facilement reconnaissable.

5. Il semble plus logique de supposer que l'affection est due à une abolition du réflexe tussigène par suite d'une irritation toxique ou réflexe, co-

[1] Communication personnelle.

incidant avec un affaiblissement des muscles respiratoires, soit immobilisation de défense, soit paralysie par névrite toxique, permettant une accumulation des sécrétions et une obstruction des bronches par ces sécrétions, provoquant l'atélectasie. Aucun de ces facteurs ne suffit à lui seul pour faire naître l'affection; il est nécessaire qu'ils coexistent.

6. Le traitement est simple et consiste à rouler le malade d'avant en arrière sur le côté sain. Il n'est souvent pas nécessaire de recourir à d'autre thérapeutique. Ce simple procédé a déterminé, dans tous les cas où l'auteur y a eu recours (cinq cas), un rétablissement rapide de l'aération pulmonaire. JACKSON et ses collaborateurs rapportent des cas de rétablissement de la perméabilité pulmonaire après plusieurs séances de bronchoscopie au cours desquelles on a débarrassé les bronches du mucus. On devra donc recourir à bronchoscopie en cas d'échec du procédé ci-dessus.

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LEGENDS

Fig. 1. Case 1. Radiograph taken March 14, 1926 — no consolidation or collapse was present at this time, but slight deviation of the trachea toward the involved side was already present.

Fig. 2. Case 1. Within thirty-six hours dense consolidation of the right upper lobe had developed. Note deviation of the trachea, but relatively slight displacement of the heart.

Fig. 3. Case 2. No definite injury. Complete collapse of the entire right lung. Note shallowness of the chest on the affected side, narrowing of the interspaces, and elevation of the diaphragm. The heart and trachea are displaced toward the affected side.

Fig. 4. Case 2. Fluoroscopic examination three days later still showed complete collapse. On rolling the patient over for the radiographic examination the lower lobe suddenly reinflated. The time which elapsed between complete consolidation and reinflation of the lower lobe was less than five minutes.

Fig. 5. Case 2. Condition three months later, showing complete recovery.

Fig. 6. Case 3. On the seventh post-operative day after a kidney fixation operation, patient complained of pain in the chest, and cough. Radiographic examation made at this time showed dense consolidation of entire left chest, approximination of the interspaces and narrowing of chest cavity, displacement of trachea and heart toward involved side.

Fig. 7. Case 3. Mucus was evacuated by bronchoscopy. No immediate result was noted. On turning the patient over on the affected side, and causing him to cough, the lung began to inflate immediately while still under our fluoroscopic vision. Note the return of the trachea almost to midline and the widening of the interspaces as inflation progresses.

Fig. 8. Case 3. Condition seven days later, showing almost complete reinflation of the left lung; small uninflated area remains in upper lobe. Mediastinum and heart have returned to their normal position. The diaphragm still remains high and is immobile.

Fig. 9. Case 4. Four days after exploratory operation: complete atelectatic collapse of the right lung; retraction of the trachea toward the involved side; pulling over of heart until it is not visible on the left side. The heart beat dislocated from its normal location was most clearly heard on the right side in the anterior axillary line. The density of the consolidation should be noted.

Fig. 10. Case 4. Three or four minutes later, without further manipulation the lung was seen to have almost completely reexpanded. The mediastinal structures have resumed their place in the midline and the heart beat is now in its normal position. The right diaphragm was still high and was immobilized. Any tendency to recollapse must be guarded against by occasional change of position of the patient for the next twenty-four hours.

Fig. 11. Case 4. Several days later the lung was still completely reinflated and the patient was free of symptoms, but the diaphragm remained immobilized in a high position.

Tabula XXIX

Fig. 2, Case I.

Fig. 4, Case II.

Fig. 1, Case I.

Fig. 3, Case II.

Fig. 5, Case II.

Fig. 6, Case III.

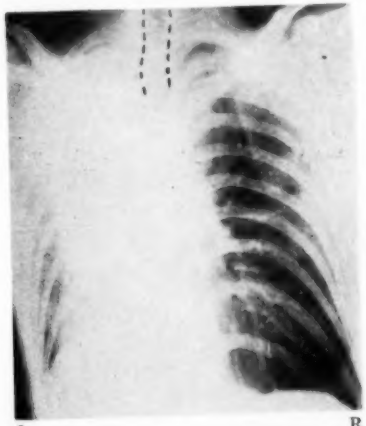


Fig. 7, Case III.

Fig. 8, Case III.

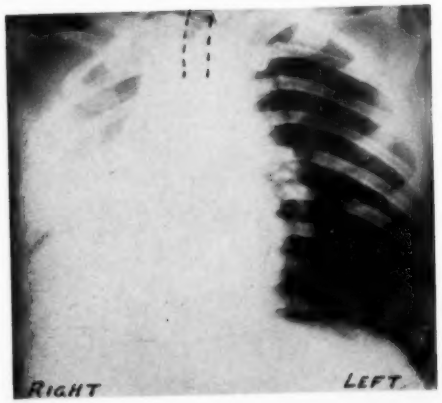


Fig. 9, Case IV.

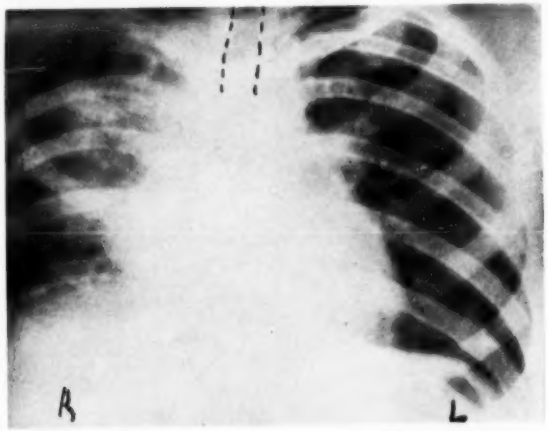


Fig. 10, Case IV.

Fig. 11, Case IV.

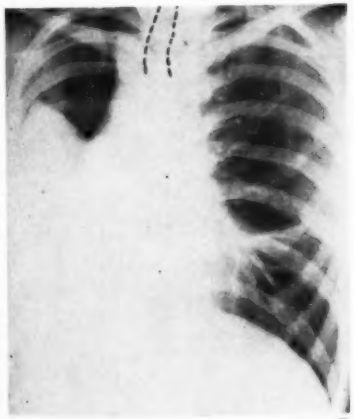


Fig. 12, Case V.

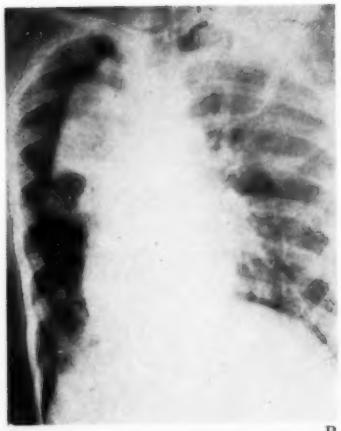


Fig. 13, Case V.

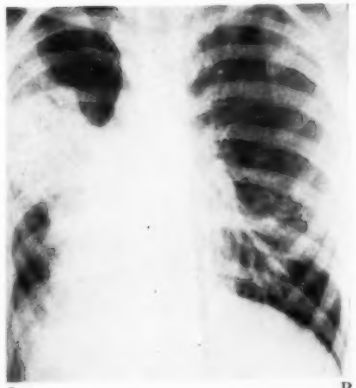


Fig. 14, Case V.

Tabula XXXI

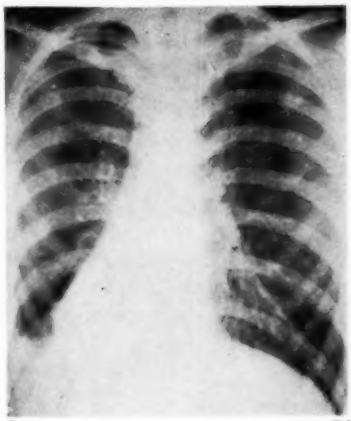


Fig. 15, Case V.

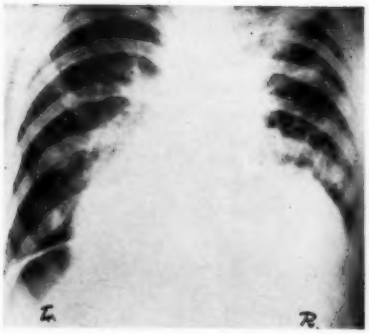


Fig. 16, Case VI.

Fig. 17, Case VI.

Fig. 18, Case VI.

Fig. 12. Case 5. (Case of Dr. HERRMAN) Massive collapse of the entire left lung with associated spontaneous pneumothorax coming on suddenly following an asthmatic attack. There is little, if any, narrowing of the chest on the involved side and the position of the ribs and interspaces are not disturbed. This is probably because the pneumothorax takes up a part of the space lost from collapse of the lung and therefore releases the tension which would otherwise be exerted against the chest wall. There is slight deviation of the mediastinum.

Fig. 13. Case 5. Rolling the patient up on his uninvolved side disclosed a rounded circular area in lower portion of the upper lobe which still was air-containing. It is possible that this small aerated area might have been unaffected by the collapse and its expansion to compensate for the loss of space incident to collapse of the remainder of the lung may have resulted in its rupture and accounted for the spontaneous pneumothorax. On assuming this position the lower lobe reinflated.

Fig. 14. Case 5. A short time later note partial reinflation and the partial remaining pneumothorax of the upper chest.

Fig. 15. Case 5. Three days later complete reinflation and absorption of pneumothorax.

Fig. 16. Case 6. Patient was struck by a train a few hours before which resulted in complete severance of the twelfth rib from its spinal attachment. The urine contained blood showing injury to the kidney on this side. Film taken at that time showed normal position of the diaphragms.

Fig. 17. Case 6. Twelve hours later, elevation and immobilization of the diaphragm was noted and a definite haziness and accentuation of the lung markings over the right lung. The process was evidently going on at this time.

Fig. 18. Case 6. Twenty-four hours after injury there was complete atelectatic collapse of the right lung.

Fig. 19. Case 6. Showing appearance after reestablishment of function. Spontaneous reinflation occurred in this case.

CONGENITAL THORACIC CYSTS AND LUNG DEFORMITIES IN THE ROENTGEN PICTURE[1]

(Communication from the Frederiksberg Hosp. Roentgen Dept. Chief: P. Flemming Møller M. D. and the Copenhagen County Hosp., Medic. Dept. Chief: Axel Jørgensen M. D.)

by

P. Flemming Møller, M. D.

(Tabulæ XXXIII—XL)

It is only rarely that the roentgenologist has occasion to diagnose a case of congenital mediastinal cyst or congenital malformation of the lung. Very few cases of the former have been published at all, and none, I believe, in which the diagnosis had been made as the result of roentgen examination. Nor do I know of any publication referring to cases of congenital pulmonary malformation diagnosed by means of roentgen.

Some interest may, therefore, attach to the fact that during the last few years I have had occasion to diagnose roentgenologically *one case of congenital mediastinal cyst* and *five cases* which were in all probability *congenital bronchiectasic, cystic formations in the lungs* — especially as the latter formations seem to present certain peculiarities interesting from the diagnostical point of view.

About two years ago, Dr. Axel Jørgensen, Senior Physician of the Copenhagen County Hospital, sent to me for roentgen examination a young man of twenty years, who, following an attack of influenza during the spring just past, had developed a very persistent cough. This had become steadily worse; and lately, when he coughed, he exhaled a breath so foul and fetid that it absolutely contaminated the air in the room. At other times there was no foul odor to his breath. He could not lie on his back, because the cough then became almost suffocating; he rested easiest when lying on his left side. His expectoration was not particularly abundant; it was not trizonal, nor did it contain any tubercle bacilli. His temperature was slightly subfebrile; otherwise he felt quite well. From his third to his seventh year he had had bronchitis every

[1] A communication laid before the Second International Congress of Radiology, Stockholm 1928.

spring. At the age of seven he had had pneumonia. In 1921 and 1923, influenza; but neither those nor the subsequent attack of that disease — in the spring of 1926 — had been attended with any pulmonary complications. Apart from this, his health had always been good.

Radiography of his thorax resulted in the surprising picture seen in *Figs. I and II*. In the left lung we observed, close up against the thorax and toward the median line, a cavity 12 × 9 cm. large, half filled with liquid. The contour of this cavity was quite sharp, and its anterior wall seemingly adherent to the anterior wall of the thorax. There was no indication of any infiltration in the surrounding tissues. The lung-tracing in every part of the left lung was perfectly sharp and natural. The heart and the vascular trunks were somewhat displaced toward the right, owing to the presence of the cavity filled with liquid. An injection of iodumbrine into the bronchi was attempted, in order to ascertain whether any passage existed between the latter and the cavity, but was unsuccessful, partly owing to the violent reflex cough which the injection excited, and which the anesthesia failed to mitigate. Only a feeble quantity got down into the bronchial ramification below the cavern, and the bronchi there appeared entirely normal. Dr. JØRGENSEN collaborating, the cyst was thereupon punctured through the anterior wall of the thorax and about 200 grams of fetid pus removed by aspiration. Immediately afterwards, 40 c. c. of iodumbrine — an oil of iodine — were injected. Almost immediately after the injection, the patient got an attack of coughing and could taste the iodumbrine in his mouth; and from the roentgen pictures taken in different positions it could be seen that a passage to the bronchi must exist, because the fine subdivisions of the latter, behind the cavity, were filled (Fig. 3). It was likewise noted, shortly after the injection, that the wall of the *left main bronchus* was coated with iodumbrine; and in a picture (Fig. 4) with the patient in a supine position there was seen, extending toward the median line, a peg-shaped projection from the shadow of the cavity which, from its situation and direction, would correspond to the left main bronchus. We, therefore, concluded that there must be a direct connection to the latter from the cyst — though probably a very tiny one, seeing that only so small a quantity of fluid passed through.

What, now, was the character of the pathological phenomenon in this case? — From the fetid odor of the expectoration it lay nearest, of course, to think of some sort of inflammation, and — under that more general symptomatic heading — considering the air- and pus-filled cavity, to conclude upon an *interlobar empyema* with perforation to the bronchi. At the same time, the situation of the cavity, upwards and anteriorly on the left side, seemed to speak against that theory, because it did not correspond to the boundaries of the interlobar fissures there.

Also the absolute clearness of the surrounding media spoke against it — as well as against any idea of a *pulmonary abscess*. To make sure that it was not a question of a *diverticulum from the esophagus*, the patient was made to swallow a few mouthfuls of baryta; but they passed through smoothly behind the cavity, thus showing that there was no communication between the latter and the esophagus. A *gravitation abscess* either from caries of a rib or from some gland was the next possibility that suggested itself; but this, it was thought, might be safely discarded. The *idea of a cysticercus* was an extremely unlikely one, seeing that the patient had never been outside Denmark, where such are never found. Roentgenologically, everything pointed rather to the case being one of an isolated bronchiectasic cavern, — or at all events of *a congenital cyst*, in which infection had set in following an attack of influenza. The subsequent operation proved this diagnosis correct.

The patient was admitted into the Municipal Hospital, Dep't V, and was operated on by Senior Surgeon, Dr. P. N. HANSEN. First, a costal resection was made, with evacuation of the pus. At the same time, a small part of the wall of the cyst was removed for the purposes of microscopy. The latter showed a thick-walled cyst, its inner surface lined with a single layer of cylindrocellular epithelium which, here and there, proliferated small glandular formations into the stroma. Underneath the epithelium, a thick layer of round cells and polynuclear leucocytes. Histological diagnosis: *Presumably an infected congenital cyst.*

The advice was for operative removal; and, consequently, a few months later, a thoracoplastic operation was made, as a preliminary measure, in order to produce the collapse of the cyst. An extensive resection was made of the second to the seventh rib. Two weeks later, the cyst was extirpated, and was found to have collapsed well; but nevertheless the cavity was immense. In some places the cyst was easy to remove; in others it adhered rather solidly to its surroundings, as, for instance, to the upper and inferior lobes of the lung, to the pericardium, and to the large vessels. It was successfully peeled out, however, so that at last it adhered only by a very small area, probably by the left bronchus; but there was no visible connection, and the matter was not explored with the probe. The cyst was extirpated, and there remained a small portion of the wall, about 2 cm. in diameter, which was cauterised, and the small surface folded up close by means of a couple of stitches. The flap of skin and muscular tissue produced by the incision was clapped back into place over the remaining cavity. The removed cyst was found to have smooth walls. *Microscopy* showed it to be congenital, of bronchious origin. In several places there were numerous glands, with high cylindrocellular epithelium; in one place there was a piece of hyaline cartilage, surrounded by mucous glands and goblet cells. Its

inner surface was lined with a single layer of cylindrocellular epithelium. Fig. 5 shows the patient after the operation.

The operation showed the cyst to be situated in the anterior mediastinum. It was, consequently, a »*congenital mediastinal cyst*». The microscopical findings suggest that it was probably the result of some congenital sequestration or forcible severance from the bronchial tree. Cysts of that kind are described in the works on pathological anatomy, and it is stated that they can arise from diverticula in the trachea as well (CHIARI). Beside those bronchogenic cysts one may find, in the thorax, *teratomata* and *dermoid cysts* arising from the anterior mediastinum. Those are the congenital cysts most frequently occurring in that locality. They may be derived from the process of thymic histogenesis; this being the case especially of the dermoid cysts. They are generally small; but they *can* grow to the size of enormous tumors which, by causing the displacement of the normal organs, give rise to severe pressure symptoms.

Clinical observations of such congenital cysts are rare. In the whole literature I have only been able to find the mention of two cases presenting the same characters as the one I am reporting here; they are both spoken of in SAUERBRUCH's: »Surgery of the Thorax».

A result of an operative treatment as fine as the present one is probably extremely unusual.

On the basis of clinical observation alone it will generally be impossible to determine exactly either from which point a mediastinal tumor arises or its character. We speak of a tumor in the anterior or posterior mediastinum; but very often tumors from the posterior mediastinum project into the anterior, or vice versa; and it happens that, even in cases where the tumor has its rise away back at the posterior aspect, the mass of it is placed anteriorly.

Among the tumors, the congenital cysts stand more or less apart. They are generally easier to diagnosticate; and they can sometimes be removed by operation, what is very rarely the case with the others.

From a roentgenological point of view it is not immediately possible, though, to conclude to the presence of a congenital cyst simply from a study of the shadow-formation in the radiograph. In the case which came under my own observation the diagnosis was not so difficult, seeing that the communication with the bronchi had allowed the passage of air into the cavity, so that the presence of a cavern half filled with liquid was immediately visible. But when such is not the case, a confusion with other pathological conditions is quite possible. In the first place, it may often be difficult to exclude the suggestion of an *aortic aneurysm*, or — if the tumor be located high up in the thorax — an aneurysm *of the anonymous artery*, as the true interpretation. Here it will be of

great assistance if a pulsation in the wall can be ascertained; but, on the whole, a comparison of the roentgen findings with the clinical observations will probably be the surest way of coming to a correct conclusion.

A *retrosternal struma* can give similar shadows in the picture. So can *genuine tumors, either of the mediastinum or the lungs.* As far as the tumors of the mediastinum are concerned, the usually lumpy, uneven contour of the shadow will be a pointer for the differential diagnosis. As regards the lung tumors, it only has to be remembered that those of which there might be question here generally occur as multiple formations, which will suffice to clear up any doubt. Still another possibility should be borne in mind, however; that, namely, *of a gravitation abscess of the anterior mediastinum* and examination should be made accordingly. An abscess of this kind has been observed by SCHINZ, arising from a caries either in the sternum or the ribs. Finally, there may be the question of *echinococcus cysts*. As these are generally situated within the lung itself, it will assist the diagnosis if an absence of communication with the mediastinum can be ascertained; but that is not always possible, and in such cases the differential diagnosis may be a matter of considerable difficulty.

Beside congenital cysts in the mediastinum, there sometimes occur *congenital cystic formations in the lung itself.* It will there be a case either of *fetal bronchiectasies* or of *cystic formations arisen in what are called »true accessory lungs»*. The former can be found diffusely scattered already through the fetal lung (MEYER, GRAWITZ), which is thus transformed into a mass of small cystic, emphysematous cavities. Where only a part of a lobe is thus affected, that part may be surrounded as by a cloak of condensed lung tissue. In other cases there may be only one large bronchiectasic cyst. Changes of that kind may either be due to malformation or may result from secretions being retained behind inflammatory bronchial stenoses. VIRCHOW and KLEBS would explain them as a distension of lymph vessels; STOERCK imagines the possibility of a cystic, fetal bronchial adenoma.

The *»true accessory lung»* — in contrast to mere supernumerary lung-lobes — have no connection with the bronchial structure, but are composed exactly like lung tissue. They are of rare occurrence. Their origin may be the result either of sequestration from the normal lung development or of a development from the fetal esophagus unconnected with the respiratory system proper; and they can, therefore, also be found below the diaphragm. Dr. CHRISTOFFERSEN, of the Frederiksberg Hospital — in a monograph on »Accessory Lungs», not yet published, but which he has kindly allowed me the privilege of perusing — states that up to about a decade ago there had been authoritatively reported in medical literature only thirty-nine cases of such formations, to which number he

himself adds two more, both observed by him at the Pathoanatomical Institute of the Copenhagen University. In one of those two, the accessory lung of a fetus was transformed into one large cyst, which displaced the lung to such an extent as almost to take its place. Other communications, as well, show the observation, in the lung, of cystic tumors that have proven to be accessory lungs. Thus, for instance, BERT-FISCHER found, in the left pleura of a woman sixty years old, a multilocular cyst as large as a fist, situated between the pleura and the diaphragm; and ROBSMANN-BEULKE found a precisely similar formation at the apex of the suprarenal gland in a man aged fifty-six, both of them proving to be accessory lungs.

If my interest has been caught by this subject, it is partly because I had occasion to diagnosticate, radiologically, the case of the congenital mediastinal cyst which I began by describing; and partly because in five other cases I have been confronted with a definitely circumscribed collection of small cystic cavities in the lung, of such a character that, on mature consideration and after exclusion of all other possibilities, I could only conclude them to be congenital bronchiectasic formations — in one case, at least, probably arisen in an accessory lung.

The *first* of those observations was made in a woman forty years old, who, in December 1925, was admitted into the Copenhagen County Hospital suffering from a severe case of *conjunctivitis*. There had never been tuberculosis in her family. She had borne four children — the last one two years ago. The process of parturition had been perfectly normal each time. She stated that »her lungs were not very strong»; but there had never been any special pulmonary symptoms, except that she often »had a cough». There had never been any hemoptysis. Though she came from a very poor home, she was in a fairly well-nourished condition. She had never suffered from night-sweats, nor had her coughing been attended with any great amount of expectoration. She had given suck to all her four children without feeling any inconvenience from so doing. It was only the conjunctivitis — an entirely uncomplicated one — that brought her into the hospital. *Stethoscopy* showed dulness in right side; anteriorly as far as costa IV, posteriorly as far as the middle of the infraspinous region of the scapula. The respiration was bronchial in the right side; and, especially after coughing, there were a great many crepitant râles and rhonchi upwards both anteriorly and posteriorly. The expectoration was very sparse, and negative as to tb. Tuberculosis being suspected nevertheless, the patient was sent to be roentgen-examined. *The radiograph* (Fig. 6) showed in the right side, in toward the hilum, a system of cavities extending from the level of the clavicle all the way down to the base. Their boundary line outwards, toward the periphery, was clearly defined, and each separate cavity was repre-

sented by a rarefaction — some of them as much as 3 cm. in diameter, — surrounded by a sharply drawn, opaque ring; the space between those rings being filled in with marked opacities. There is liquid in the bottom of all the rarefactions. With the patient lying on her right side it is seen that the surfaces of this liquid have shifted, which proves that it is really a case of large cavities. There are adhesions downward to the dome of the diaphragm. The lung tissue which enfolds the system of cavities as with a cloak presents nothing decidedly abnormal. The right apex and the upper part of the right thorax are more barrel-shaped dilated than the left ones, and there is a displacement of the heart toward the left. In *a side-view* (Fig. 7) it can be distinctly seen how the system of cavities occupies a well-defined portion of the lung, lying downward from and a little forward of the hilum, but at the same time with its upper portion extending somewhat posteriorly. The left lung shows nothing abnormal, except for a calcified focus in its lower part.

The patient absolutely refused to remain any longer in the hospital for further examination, declaring that »there was nothing the matter with her lungs». Under those circumstances, a planned examination by means of iodumbrine had to be adandoned, and since her discharge all attempts to get into renewed communication with her have failed. All the facts that *were* ascertained, however, — the evident presence of an irritant factor in her lungs ever since childhood, her own statement that »her lungs were not so very strong», and the curious system of cavities sharply limited to one particular portion of the lung without any positively demonstrable infiltrations into any part of the surrounding tissue — point strongly to the conclusion that this pathological condition has not developed on the basis of inflammatory changes, but was in all probability of congenital origin. Then, assuming this to be the case, it lies very near, in my opinion — seeing that the changes were so definitely circumscribed — to think of them as cystic changes in an accessory lung; especially as the very slight amount of expectoration seems to indicate that no communication exists between the large cavities and the bronchi. Still, it is, of course, impossible to say anything absolutely definite on this point; especially as there was no opportunity for making an injection of iodumbrine into the bronchi.

The *second case* resembles the first one in a great many ways, both clinically and in the radiograph. It was that of a woman, twenty-four years old, admitted into the tuberculosis ward of the Frederiksberg Hospital in the spring of the year 1927. As a child she had never been ill. She had borne three children and had nursed them all. In the summer of 1919, when she applied for admission into a sick-club, »something» was discovered in her left lung. She, therefore, had herself admitted into the Oeresunds-Hospital, and remained under observation there for

one month. Examinations proved her to be free from tubercle bacilli. Since then, two of her children have been born, and she is feeling in excellent health. Four weeks before her recent admission into the Frederiksberg Hospital she came to the Tuberculosis Station to have one of her children examined, and it was on that occasion that — after she had herself been examined also — she was advised to seek admission into the hospital once more. She neither coughs nor expectorates, she feels perfectly well, and there is nothing in her condition that answers the symptoms of a tuberculosis. *Stethoscopical examination* showed very little out of the ordinary. There was a slight dulness and a very few râles above the right apex, with slightly bronchial respiration. Tubercle bacilli could not be proven. The blood-sedimentation test varied between 6 and 12 mm. She was pregnant when she came to the hospital, and has given birth to a child since.

The *radiographic picture* (Fig. 8) is extraordinary. The upper third of the right lung is completely taken up by a system of cavities, the boundaries of which encroach on one another in a perfectly disordered manner. There is no collection of liquid in any of those cavities. Their separation from the surrounding, to all appearances healthy lung tissue is not so sharply defined as in the other case just described; but it seems as if the cavities filled the whole of the superior lobe (Fig. 9).

There are calcifications in the hilum, especially in the left side; but otherwise the roentgen picture reveals no abnormal features. There is no shrinking in the right lung proportionate to the extensive pathological changes there. In the hope of getting some more light on the actual character of the system of cavities an injection of iodumbrine was attempted, but unsuccessfully owing to the coughing of the patient.

An absolutely positive diagnosis of this case would seem to me a hazardous venture. The possibility that it may be a case of tubercular caverns, or of bronchiectasies resulting from shrinkings attendant on a tuberculous affection, cannot be excluded. At the same time, the picture does not — either roentgenologically or clinically — resemble a case of true tuberculosis. Nor do I know of any other kind of pulmonary affection ever observed, that would explain the occurrence of a system of cavities like the ones in this case. There remains, then, also here the possibility of a congenital affection. The fact that no liquid is observed in the cavities may possibly indicate that they have been drained; and that, again, may suggest that in the present case we have to do — not, as in the previous case, with cystic changes in an accessory lung, but rather with a case of fetal bronchiectasies.

The anamnesis in the three following cases is exactly the same as in the two already described. There had been a slight tendency to coughs, but never any actual affection of the lungs, and a thorough examination

by all available methods failed to give any substantial grounds for suspecting tuberculosis. Like in the first case, the roentgen examination here gave a picture absolutely surprising in view of the stethoscopy and the previous history of the patients.

The *third case* was that of a married woman, 31 years old, who was admitted into the Copenhagen County Hospital for a metrorrhagia. She had several times had catarrhal bronchitis, but never pneumonia; and there was no disposition to tuberculosis in her family. She had given birth, in the normal way, to six children, who are living and in good health; and she has aborted twice, the last abortion, three years ago, leaving the endometritis for which she has now entered the hospital. — Microscopy of scraped-out mucous membrane showed acute inflammation, but no signs of tuberculosis.

She has for many years had a cough, to which she doesn't attach any importance. There has been no night-sweat, no stitch, no loss of weight; and there has never been found tubercle bacilli in her sputa.

Stethoscopy: M_1 around left apex to C_1 anteriorly; M_2 to angle of scapula posteriorly; M_1 to base. Bronchial respiration over the dull area. Below the scapula sharp bronchial respiration, bronchophony and exaggerated vocal fremitus. Nowhere râles, even after coughing. The narcosis was difficult, because she had very little narcotic latitude and very easily became cyanotic. As pulmonary tuberculosis is suspected, she is sent to be roentgen-examined. During her stay in the hospital there was no expectoration, nor did her temperature go above the normal.

Roentgen Examination of the thorax revealed a curious condition of the left lung (Fig. X). The whole of that organ is changed into a system of larger and smaller cavities surrounded by reticular consolidations, with no pulmonary tissue between them. The cavities are largest upwards in the lung, and do not seem to contain any fluid. The whole left half of the thorax is very much shrunk, just as the lung itself. The mediastinum and heart are drawn away over towards the left, and a *side view* (Fig. XI) shows that the heart is also drawn strongly backwards. There is probably no normal pulmonary tissue at all in the left lung.

It is difficult to make an absolute diagnosis; but also in this instance it is very unlikely that the case should be one of tuberculosis with cavernous formations. For a moment, the idea of a diaphragmatic hernia suggested itself, because the cavities in the upper part might look rather like loops of the small intestine; but then, on the other hand, the diaphragm could distinctly be traced, unbroken, downwards above the plainly visible gas-filled sac of the stomach.

The most likely conclusion is that, in this case also, we are in the presence of a *congenital affection* involving a cystic alteration of the

whole lung, the cavities in the latter being in the nature of *congenital bronchiectasies* — in other words, what is commonly known as the »*honeycomb lung*». There is nothing abnormal about the right lung.

The *fourth case* was that of a young girl, 19 years old, of a healthy family. She had always been, well up to 1922, when she was admitted into the Kolding Sanatorium as suffering from coughs, lassitude and loss of flesh, following an attack of the grippe without any special complications. Her sputa were repeatedly examined for tubercle bacilli, but the result was always negative. Tuberculin reaction negative. Moro's test negative. As the sense of weariness continued, and she did not have any appetite, she was admitted, on April 8th, 1924, to the Oeresunds Hospital, where her case was diagnosed as pulmonary tuberculosis of the 3rd degree. *Stethoscopy on admission*: *Right* lung: M_1 over the anterior surface to C_2, with bronchial respiration and a few râles; M_1 over posterior surface to $^1/_2$ Sc., with weakened bronchial respiration, râles and rhonchi. *Left* lung: M_1 to C_3 and just below the spine of the scapula. Bronchial respiration anteriorly; weakened bronchial respiration posteriorly, with a few râles. *Stethoscopy at the time of discharge*, July 24th, 1924: No râles in the left side; much fewer râles in the right side. — Her temperature was normal during her whole stay in the hospital. Blood-sedimentation, 1 hour: 3. Hemoglobin, 80 per cent.

She was not roentgen-examined at the time. Since her discharge she has been in good health; but as she still continued to cough a little, her physician sent her to my private roentgen clinic for examination, in the spring of 1927, with the following result:

Roentgen Examination (Fig. XII) showed, in the right side, inwards toward the hilum, a number of cavities, of various size, lying close to one another, without any pulmonary tissue between them. They are strictly localised to this portion of the lung, and each of them is sharply circumscribed from the others. Except for some slight calcification in the neighbourhood of the hilum, the surroundings are perfectly normal, without any signs of either old or recent infiltrations; and there are no shrinkings in the lung. *In the side-view* (Fig. XIII) the cavities are seen situate, sharply circumscribed, anteriorly from the hilum. The left lung is normal.

The changes are precisely similar to the ones already described. Nor can there, in my opinion, be any doubt but that, in this case also, they are congenital, bronchiectasic malformations. There is nothing in the roentgen picture hinting at tuberculosis; nor does the case bear any resemblance, clinically, to that disease; nor were there at any time during the earlier hospitalisation of the patient observed any signs that would justify a positive diagnosis to that effect.

The *fifth case* was that of a married woman, 45 years old, admitted into the Frederiksberg Hospital, on June 16th, 1928, for a hemoptysis.

No family disposition to tuberculosis. No scrofula in her young age; influenza in 1918, without complications. Otherwise she has always been in good health, except for a tendency to coughs when she was a child; and the only time she has consulted a physician was when she had the attack of influenza just mentioned. Her cough has not been getting worse lately; she has always expectorated a little. During the last six months she has been feeling tired, and has lost about 8 pounds in weight. Night-sweats now and then; she coughs and expectorates a good deal. There had never been any blood in her sputa until June 15th, 1928, when she brought up some bloody splotches; but there was no considerable hemoptysis. She is rather thin, but not particularly enfeebled. On her left cheek, and extending over the left side of her nose, there is a hemangioma about as large as the palm of a child.

During the beginning of her stay in the hospital she brought up about 40 ccm. of bloody sputum, which has a tendency to be trizonal, but which is not fetid.

Stethoscopy of the lung: Dulness in left side from spine of the scapula to base; with respiration very faint (inaudible downwards); no râles. Over the clavicle likewise bronchial respiration, with few râles. Anteriorly, downwards, respiration very weak. — Laryngoscopy showed normal conditions. No tubercle bacilli in the sputa. Blood sedimentation: June 21st. 98 mm.; August 4th. 38 mm. — Hemoglobin, 10.3 per cent.

The *roentgen picture* (Fig. XIV), curiously enough, is an almost complete replica of the picture presented by Case III.

In the upper portion of the left lung is seen a system of cavities, large and small, without any pulmonary tissue between them, their outlines forming an irregular net-work pattern. In the apex and below the clavicle, out towards the periphery, there is nothing except those cavities; but downwards from there begins a diffuse, intense consolidation, which extends all the way to the base, where it merges with the shadows of the diaphragm and heart. Even through this diffuse consolidation there can be seen, however, away down in the inferior portion of the lung, several distinctly circumscribed rarefications, indicating the existence of cavities also in that portion of the organ.

The whole left lung, as well as the left half of the thorax, are very much shrunk; with the result that the heart is drawn away over to the left side and cannot be seen at all through the consolidations. Also the trachea and the bifurcation are displaced towards the left. There does not appear to be any fluid in the cavities.

Also in *the side-view* (Fig. XV) the cavities are plainly seen in the upper part of the organ, and it seems as if the heart is perhaps displaced

somewhat posteriorly in the thoracic cavity. Just as in the antero-posterior view, the left diaphragm appears to be situated rather high up; and immediately below it is seen the gas-filled stomach.

The roentgenological picture does not look like one of tuberculosis. At the same time, it cannot be denied that it *may* be an old fibrous phthisis with cavern formations. The anamnesis of the case and its roentgenological peculiarities nevertheless make the suggestion highly improbable, and the most likely interpretation seems to be that of a *congenital anomaly* involving the whole of the left lung, with cystic bronchiectasic formations in the upper portion of that organ, and an atelectatic condition farther down. The cavities in the upper part do not look like tubercular excavations, and the enormously shrunken condition of the whole lung likewise points to a congenital malformation — something in the nature of a »honeycomb-lung», just as in Case III.

The hemoptysis may be due to bleeding from the bronchiectasies; but it may also be due to a beginning pathological change — the possibility of a malignant degenerative process being perhaps the nearest suggestion.

In the right lung there is noticed a vicarious emphysema, but otherwise nothing abnormal.

I cannot bring any positive proof to show that these five last cases are actually congenital malformations of the lungs; but, everything considered, I believe that diagnosis to be the most likely one. In every one of the cases the roentgenological picture is highly interesting. It lies very near, of course, to mistake them for cases of cavernous tuberculosis; but the remarkable fewness of clinical symptoms, taken together with the surprising roentgenological findings — which contrast so sharply with the otherwise normal roentgenological picture — answers very well to what one would expect to find in the case of such congenital affections. An interesting point for the diagnosis is furnished by the peculiar way in which the cavities are situated in relation to one another: sharply delimited by fine, reticulated consolidations, without any infiltrations or pulmonary tissue between them, and forming a tangle without the slightest apparent regularity. Such pictures will probably never be found in cases of tuberculous changes.

If sufficient attention is paid to features such as the ones here described, it may perhaps be found that congenital malformations of the lungs are not so very rare. At all events, it is only by means of the roentgen examination that they can be detected during life.

SUMMARY

The author reports a case of congenital mediastinal cyst, diagnosed by means of roentgen examination and subsequently healed as the result of operation. In that connexion he discusses the question of congenital cystic formations in the lung, and describes five cases of congenital bronchiectasic, cystic formations in that organ, likewise diagnosed roentgenologically; one of them probably developed from an accessory lung. He remarks on the curious, characteristic roentgen picture resulting in those five cases — four of which were incidental findings — and points out how easily it may be mistakenly interpreted as representing a cavernous tuberculosis. The diagnosis of this congenital malformation, which has not been roentgenologically described before, will not be difficult, however, when the extreme paucity of clinical symptoms is taken in connexion with the characteristic roentgenological features, the most notable one of which is the peculiar disposition of the cavities in relation to one another; irregularly arranged, each one sharply delimited and separated from the others by a fine network of consolidations, without any infiltrations or pulmonary tissue in between.

ZUSAMMENFASSUNG

Verf. berichtet über einen Fall von kongenitaler Mediastinalzyste, die mittels Röntgenuntersuchung diagnostiziert wurde und später nach einer Operation heilte. Er erörtert in diesem Zusammenhang die Frage kongenitaler Zystengebilde in der Lunge und beschreibt 5 Fälle von kongenitaler bronchickatischzystischen Formationen in diesem Organ, die gleichfalls röntgenologisch diagnostiziert worden waren; eine von ihnen hatte sich wahrscheinlich aus einer akzessorischen Lunge entwickelt. Er macht auf das merkwürdige charakteristische Röntgenbild aufmerksam, das sich in diesen fünf Fällen ergibt — vier von ihnen waren zufällige Befunde — und betont, wie leicht sie fälschlich als kaverhöse Tuberkulose ausgelegt werden können. Die Diagnose dieser kongenitalen Missbildung, die bisher nicht röntgenologisch beschrieben worden ist, wird indes nicht schwer sein, wenn die äusserst geringen klinischen Symptome zusammen mit den charakteristischen röntgenologischen Zügen in Betracht gezogen werden, von welchen der auffallendste die eigentümliche Disposition der Hohlräume zu einander ist, die unregelmässig angeordnet, je für sich scharf begrenzt und von den anderen durch ein feines Netzwerk von Verdichtungen getrennt ist, ohne jegliche Infiltrationen oder Lungengewebe zwischen ihnen.

RÉSUMÉ

L'auteur communique un cas de kyste congénital du médiastin, dont le diagnostic a été fait par l'examen radiologique et qui a été ultérieurement guéri par une intervention opératoire. Il discute à ce propos la question des formations kystiques congénitales du poumon et décrit cinq cas de formations kystiques et bronchiectasiques congénitales de cet organe, également découvertes par l'examen radiologique et dont l'une était probablement développée

Fig. 2.

Fig. 1.

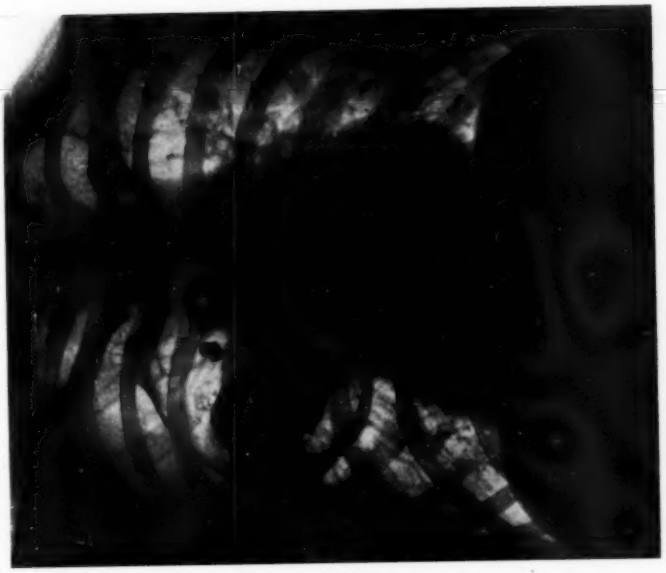


Fig. 4.

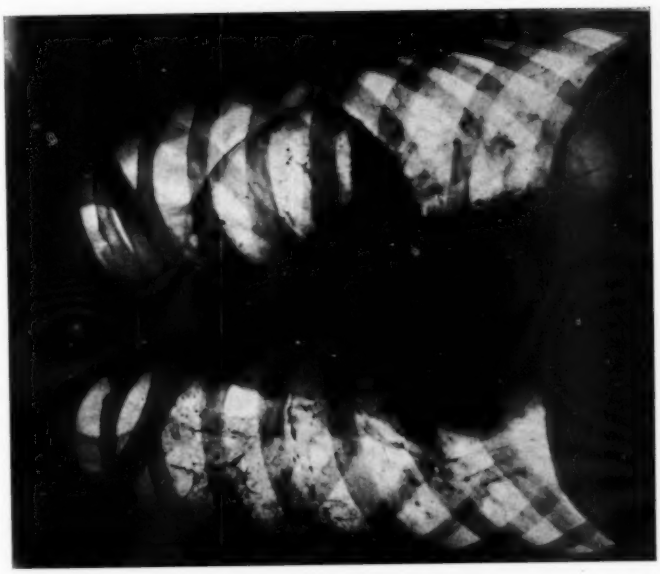
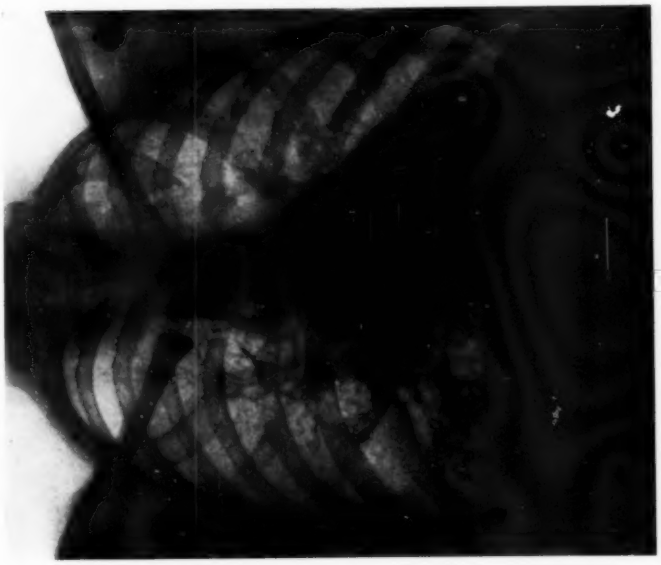


Fig. 3.



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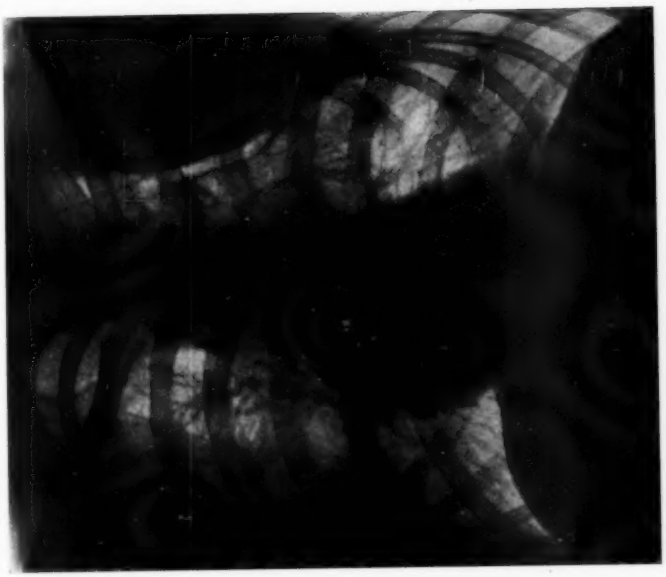

Fig. 5.

Fig. 8.

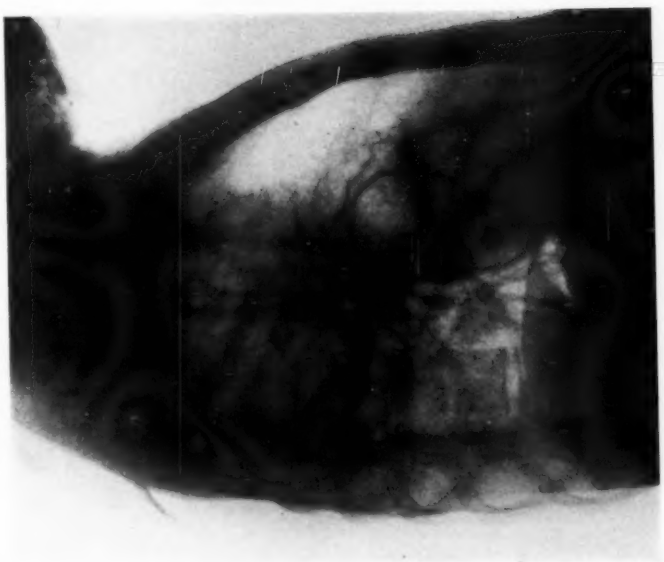

Fig. 7.

Fig. 10.

Fig. 9.

Fig. 12.

Fig. 11.

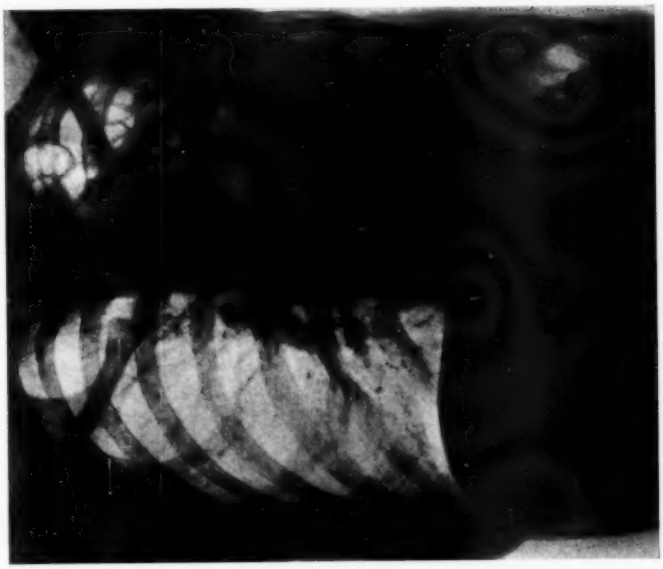

Fig. 14.

Fig. 13.

Fig. 15.

aux dépens d'un poumon accessoire. L'auteur attire l'attention sur l'aspect curieux et caractéristique de l'image radiologique donnée par ces cinq cas (dont quatre furent découverts d'une façon purement fortuite), et fait observer la facilité avec laquelle ils peuvent en imposer pour une tuberculose caverneuse. Le diagnostic de cette malformation congénitale du poumon, qui n'a été antérieurement l'objet d'aucune description radiologique, ne saurait présenter de difficultés si l'on considère l'absence à peu près complète de signes cliniques comparée à l'aspect caractéristique de l'image radiologique; l'un des points les plus notables de cette image consiste dans la disposition particulière qu'affectent entre elles les cavités, irrégulièrement distribuées, nettement délimitées et séparées les unes des autres par un fin réseau d'épaississements, sans aucune infiltration du tissu pulmonaire adjacent.

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THERMOGENESIS BY RADIO FREQUENCY CURRENTS

A PRELIMINARY ANNOUNCEMENT

Presented before Section D, 2nd International Congress of Radiology, Stockholm July 25, 1928

by

Albert Soiland, M. D., Los Angeles

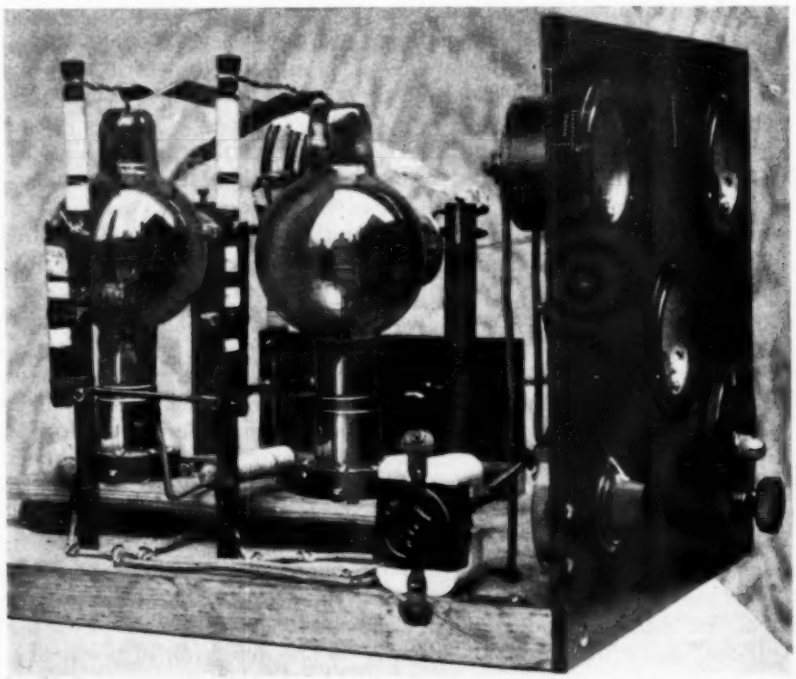
Some time ago our associate, Dr. ORVILLE N. MELAND, became imbued with the idea that inductive heat, such as that observed in workers around a radio broadcasting station, might be of therapeutic value. This thought was amplified when applied to the well-known fact that heat may destroy cancer cells; but the heat that has heretofore been employed for this purpose has been of the conductive variety, that is, actual heat applied to the surface through any means which generates this, and also from the use of various surgical types of cautery. All medical men are familiar with the limitations of surgical heat except when directed to a cancerous lesion which is of strictly local character. For this reason most modern clinical centers have discontinued surgical heat as a major appliance. Medical heat, however, such as hot packs, therapeutic lamps of all sorts, and medical diathermy apparatus, is rising in favor. These depend upon the conductive factors of heat distribution where this is distributed to various parts and depths of the body, according to the amount of energy in the heat-producing source. None of these have as yet demonstrated their ability to carry sufficient heat into the parts to destroy cancer cells without being used in amounts dangerous to the life of the patient. Recent observations by MENDEL and WALINSKI, reported in the »Klinische Wochenschrift» and abstracted in the Journal of the American Medical Association of May 19th, 1928, give an interesting account of their experience with hot packs on cancer and tumor cells.

The fever heat, such as that obtained by inoculation of various proteins and toxins, will not be referred to here, as this belongs to a very important but different line of investigation.

This leads us up to the subject of inductive heat, a term which the writers take the liberty to use, and as produced by radio-frequency currents.

In various popular science magazines, instruments have been described for generating radio-wave energy, and these may be employed

to induce heat in the patient's body. This heat can be inducted into the interior of the patient's body without increasing the skin temperature more than that of the internal parts, and without the use of contact electrodes. This is the type of apparatus which we are now investigating and have initiated treatment along experimental lines, and of which we desire to make a brief preliminary report.



Radio frequency set with control board.

With the assistance of Dr. ARTHUR H. WARNER, a physicist connected with the California Institute of Technology, and Mr. GILBERT WRIGHT, a skilled young scientist interested in radiophysics, two instruments have been constructed, the first a crudely designed affair with which we were able to raise the patient's temperature from normal to 1.5 degrees plus Fahrenheit. The second instrument, a photograph of which is herewith submitted, has been much better assembled and improvements in winding perfected so that a higher temperature rating may be obtained. In Mr. WRIGHT's own words, the set-up of the apparatus is as follows:

»The arrangement of apparatus giving the best results to date is essentially a radio-transmitting circuit operating with fair power (uρ to one thousand watts) and at wave lengths of from three to ten meters. This corresponds to frequencies of from one hundred million to thirty million cycles per second. Currents of these frequencies are produced in the usual manner and are fed into a condenser. It is this condenser which is used as an applicator to induce heat through a portion of the patient's body. The condenser consists of two protectively insulated copper foil plates and the tissues of the patient. From an electrical standpoint such a condenser is a poor one owing to the inferior dialectric properties of protoplasm which cause much of the electrostatic energy of the condenser to be dissipated as heat.

»Although this heat can only be felt by the patient at the skin surfaces the heat intensity should be uniform through the entire mass of tissue between the plates. These plates may be of any practical size or shape and can successfully induce heat through any desired thickness of flesh included between them. There is absolutely no danger of electrical shock to the patient in a properly designed circuit and the heat generated can have any desired value and may be regulated with any degree of minuteness.

»Considerable work has yet to be done with the electric circuits before the apparatus can be made to give efficient performance as a heat generator. This is the work of the competent radio engineer and even for him, accustomed as he is to radiofrequency phenomena, this field of frequency between the infra-red and thirty million cycles per second is relatively a new one. But in connection with this form of heat induction and the attendant molecular effects due to these unusual electrical frequencies, quite aside from their heating properties, there are far greater problems. The solution of these problems must be the work of the physicist, the chemist, the biologist, and above all, the capable and ethical physician.«

Our reason for bringing this so early to the attention of the medical profession is with a realization that here is a potential agent which, by means of insensible radio-frequency waves, may induce physical changes as heretofore not recorded; may have a singular bearing upon certain metabolic disease, and it is hoped may give us a new and additional weapon of offence against cancer.

The all-important reason for this announcement, however, is to sound a serious note of warning against the general use of this easily obtained apparatus as a means of exploiting the public, and to plead for some sort of medical supervision for this potent and as yet little understood therapeutic agent.

A few clinical notes from observations of Doctors Costolow and Meland are appended:

Locally:

Relief of pain and production of localized heat where pads are adjusted.

Generally:

(1) Sensation of general body warmth, accompanied by a profuse perspiration.

(2) Rise of body temperature from 5 to 1.5 degrees Fahrenheit.

(3) Soothing effect on the nervous system, leading to a restful sleep.

Besides these general effects on the patient, there is a sense of depression that comes over the attendant when he works around the tubes too long. This may go on to the point where there is a continuous dull headache.

Clinically we have used it in cases of arthritis, where it seems to work better than ordinary diathermy.

SUMMARY

The author gives a preliminary report of using, as a therapeutic agent, the inductive heat as produced by radio-frequency currents. From the description of the apparatus is found that the transmitting wavelengths can be varied from three to ten meters, corresponding to frequencies of from one hundred million to thirty million cycles per second. The transmitting circuit is operated with a maximum power of 1 000 watts. A rise of body temperature from 5 to 1.5 degrees Fahrenheit has been obtained. The author expects here to find a potential agent which may give an additional weapon of offence against cancer.

ZUSAMMENFASSUNG

Verf. macht eine vorläufige Mitteilung über die Verwendung der durch hochfrequente Radioströme erzeugten »induktiven» Wärme zu therapeutischen Zwecken. Aus der Beschreibung des Apparates geht hervor, dass die Senderwellenlängen zwischen 3 und 10 Metern variiert werden können, was Frequenzen von 100,000—30,000 Kc/s per Sekunde entspricht. Es wurde mit einer Maximalleistung des Senderstromes von 1,000 Watt gearbeitet. Dabei erreichte man Steigerung der Körpertemperatur zwischen 5 bis 1.5 Grad Fahrenheit. Verf. glaubt, hier ein wirksames Agens zu finden, das als eine weitere Waffe zur Bekämpfung des Karzinoms beitragen mag.

RÉSUMÉ

L'auteur donne un aperçu préliminaire de l'usage, comme agent thérapeutique, de la chaleur inductive produite par la radio-courants de fréquence. De la description de l'appareil, il ressort que la longueur d'onde de transmission peut être variée de trois à dix mètres, correspondant à des fréquences allant de cent millions à trente millions de périodes par seconde. Le circuit de transmission fonctionne avec une énergie maxima de 1000 watts. On obtient une augmentation de la température du corps de 5 à 1.5 degrés Fahrenheit. L'auteur espère trouver ici un agent puissant, susceptible de fournir une arme nouvelle dans la lutte contre le cancer.

AUS DEM AKADEMISCHEN KRANKENHAUS ZU UPSALA

DER MANOMETER, EIN HILFSMITTEL ZUR ERZIELUNG SCHARFER RÖNTGENBILDER RESPIRATORISCH BEWEGLICHER ORGANE

von

Hugo Laurell

Bei der Röntgenaufnahme respiratorisch beweglicher Organe in der Brust- und Bauchhöhle ist es, wenn man technisch gute Bilder haben will, natürlich oft wichtig, die Belichtung bei völligem Atemstillstand zu machen, da das Bild sonst mehr oder weniger unscharf werden kann. Man braucht sich nur daran zu erinnern, dass gewisse Gallensteine, die auf scharfen Bildern wie Ringschatten aussehen, auf anderen Aufnahmen, die unabsichtlich während einer anhaltenden, wenn auch schwachen Atmungsbewegung aufgenommen sind, unsichtbar sein können. Ja, schon eine ganz geringe Verschiebung des Zwerchfells während der Belichtung kann in gewissen Fällen genügen, wichtige Details, wie Ringschatten bei Gallensteinen, auf dem Röntgenbild verwischt und undeutbar zu machen.

Der Atmungszustand ist natürlich besonders wichtig in solchen Fällen, wo die Zeit der Belichtung aus dem einen oder anderen Grunde verhältnismässig lang ist. Es genügt in der Regel nicht, den Patienten unmittelbar vor der Belichtung aufzufordern, den Atem eine gewisse Anzahl Sekunden anzuhalten, man muss ihn vielmehr unter genauer Kontrolle sich hierin üben lassen. Eine derartige effektive Kontrolle ohne besondere Hilfsmittel durchzuführen, ist jedoch schwierig.

Als ein Mittel, um den Atmungszustand des Patienten zu kontrollieren, ist seit 1926 im Akademischen Krankenhaus ein gewöhnlicher mit Skala und Schlauch versehener Wassermanometer mit gutem Erfolg benutzt worden (Fig. 1). Der Schlauch, in den der Patient hineinblasen oder an dem er saugen muss, ist zu diesem Zweck mit einem leicht austauschbaren, einfachen Mundstück aus einem Stückchen Glasrohr versehen. Die Skala befindet sich zwischen den beiden Schenkeln des Manometers und ist daselbst an beiden Seiten in cm. Wasser gradiert. Die Flüssigkeitssäule hat einen Durchmesser von ca. 5 mm., und die Flüssig-

keit ist mit roter Tinte gefärbt, sodass die Stellung des Flüssigkeitsniveaus auch aus einiger Entfernung leicht abgelesen werden kann. Die eine Seite der Skala liest der Patient ab, die andere gleichzeitig der Untersuchende, der auch kontrolliert, dass der Patient das Flüssigkeitsniveau auf einem angegebenen Plus- oder Minuswert hält. Man stellt den Manometer, wenn er benutzt werden soll, auf einen kleinen Tisch vor den Patienten und wendet seine Skala so, dass sie teils vom Patienten teils von dem Platz des Photographen aus abgelesen werden kann. In der Regel wird der Patient aufgefordert, nach einer tiefen Einatmung langsam und vorsichtig das Flüssigkeitsniveau bis zu dem Zeichen hinaufzublasen, das einen Plusdruck von 10 cm. Wasser angibt, und es dort 5—10 Sekunden so still wie möglich zu halten; während dieser Zeit geschieht dann die Belichtung. Für gewisse Zwecke ist es manchmal notwendig, einen etwas grösseren Druck anzuwenden, z. B. in solchen Fällen, wo die äusseren Kontraktionen des Herzens so lebhaft sind, dass die Lungenstruktur nach dem Herzen zu auf dem Röntgenbild unscharf wird. Dann geschieht die Belichtung erst, nachdem der Patient den Atem 10—15 Sekunden während eines Plusdruckes von 10—20 cm. Wasser angehalten hat. Durch diese in ihrem Effekt einem *Valsalva*'schen Versuch entsprechende Massregel nimmt das Herz an Breite ab und seine Pulsationen werden geringer. Dieser Versuch ist für den Patienten etwas anstrengend und darf nicht angewandt werden, wenn schwere Herzfehler vorliegen. In anderen Fällen kann es hingegen zwecks Erforschung gewisser Spezialfragen in Bezug auf die Blutverteilung in den Lungen von Interesse sein, die Lungenbilder während eines geringen intraalveolären Unterdruckes aufzunehmen.

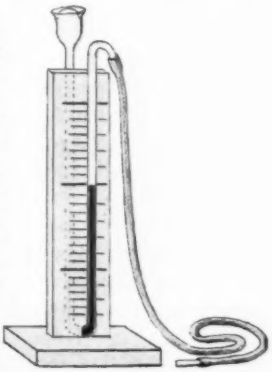


Fig. 1.

Untersucht man bei ein und demselben Patienten die Lungen zu wiederholten Malen, so muss derselbe intraalveoläre Druck bei den verschiedenen Untersuchungen angewandt werden, sodass sich die Bilder so weit wie möglich vergleichen lassen.

Was die Fernphotographie des Herzens mit Manometerkontrolle anbelangt, so muss man in Betracht ziehen, dass die Grösse des Herzens bei verschiedenem intraalveolärem Druck und verschiedener Zwerchfellstellung erheblich wechseln kann. Es ist daher wichtig, dass man sich immer derselben Technik bedient, am besten vielleicht eines geringen Plusdruckes, da es für viele Personen leichter ist, bei einem geringen Plusdruck das Flüssigkeitsniveau still zu halten als bei einem geringen Minusdruck. Ferner muss die Belichtung nach einer bestimmten Anzahl

Sekunden und wenn möglich bei derselben Zwerchfellstellung (am besten Anhalten des Atems nach einer schwachen Einatmung) geschehen.

Wenn der Manometer benutzt wird, so muss seine Luftsäule mit der Lungenluft in offener Verbindung stehen; es gelingt dabei nie, das Flüssigkeitsniveau bei einem gewissen Plus- oder Minus-zeichen auf der Skala ganz still zu halten, denn die pulsatorischen Druckveränderungen, die normaliter in der Lungenluft durch die Herztätigkeit hervorgerufen werden, und die sich in dem s. g. pneumatischen Puls zu erkennen geben, beeinflussen offenbar die Flüssigkeitssäule, sodass sie um eine Gleichgewichtslage deutliche pulsatorische Oszillationen ausführt, die an Amplitude von einigen mm. bis zu ca. einem cm. wechseln. Steht die Flüssigkeitssäule während eines Versuches einmal vollkommen still, so weiss man, dass er falsch ausgeführt wird, denn dann ist ein Hindernis für die Luftpassage vorhanden, und der Manometer erfüllt nicht seine Aufgabe als Kontrollapparat. Die Ursache für das Stillstehen der Flüssigkeit ist in der Regel leicht zu finden; es kann z. B. der Schlauch vorübergehend geknickt sein, oder auch verschliesst der Patient das Mundstück mit der Zunge.

Der Patient lernt in der Regel leicht nach Instruktion und kurzer Übung, das Flüssigkeitsniveau bis auf einen cm. still zu halten. Bei der Übung wird er in der Regel aufgefordert, einen tiefen Atemzug zu tun, und dann das Flüssigkeitsniveau bei einem Zeichen zu halten, das einem schwachen positiven Druck entspricht. Fast ausnahmslos gelingt es ihm auch später während der Belichtung die Flüssigkeitssäule so still zu halten, dass das Röntgenbild des Zwerchfells und der Lungen scharf wird. Auch wenn die Flüssigkeitssäule während der Belichtung einige cm. fluktuieren sollte, so zeigt das Zwerchfell scharfe Konturen und das Bild der respiratorisch verschiebbaren Organe wird in der Regel völlig befriedigend. Es ist nämlich eine erhebliche Verschiebung des Flüssigkeitsniveaus während der Belichtung erforderlich, damit das Zwerchfell sich mit einer auffallend unscharfen Kontur zeichnet, was offenbar damit zusammenhängt, dass der Manometer im Verhältnis zu der Thoraxkavität eine so geringe Weite hat.

Man erhält, wie gesagt, fast ausnahmslos mit Hilfe des Manometers sofort scharfe Bilder; in der Regel auch in solchen Fällen, wo kurz zuvor wiederholte Bilder, auf die gewöhnliche Weise aufgenommen, misslungen worden sind, weil der Patient den Atem nicht anhalten konnte. Mehrere Ursachen tragen zu diesem günstigen Resultat bei. Die Übung bei genauer Kontrolle erhöht in hohem Grade die Fähigkeit des Patienten, den Atem anzuhalten. Der Untersuchende macht nie eine Belichtung, ehe er sieht, dass der Patient eine gewisse Übung erhalten hat. Indem man acht gibt, wieviele Sekunden für den Patienten erforderlich sind, das Flüssigkeitsniveau bei dem gewünschten Skalastrich einzustellen,

kann man den richtigen Zeitpunkt für die Belichtung wählen. Vor und während der Belichtung muss der Patient seine Aufmerksamkeit darauf konzentrieren, die Flüssigkeitssäule still zu halten, weshalb er nicht so wie sonst die Laut- und Lichtphänomene, die mit der Belichtung verbunden sind, merkt und nicht so sehr durch sie beunruhigt wird.

Der Manometer ist im Akademischen Krankenhaus namentlich beim Photographieren von Lungen benutzt worden (diese werden hier häufig in einem Fokus-Plattenabstand von 1.5—2 Metern und mit einer Belichtung von 1—1.5 Sek. aufgenommen), ferner bei der Aufnahme von Gallensteinen und Nieren. Dass er auch in andern Fällen von Nutzen sein kann, geht aus dem oben Gesagten hervor.

ZUSAMMENFASSUNG

Verf. weist nach, dass man häufig gute Hilfe von einem Wassermanometer hat, wenn man von respiratorisch beweglichen Organen wie Lungen, Nieren und Gallenblase, scharfe Röntgenbilder haben will, Momentaufnahmen aber nicht fertigen kann.

SUMMARY

The author shows one is often greatly helped by a water manometer when wishing to obtain sharply defined radiograms of organs moving with the respiration, such as lungs, kidneys and gall-bladder.

RÉSUMÉ

L'auteur démontre que le manomètre à eau est souvent d'un secours précieux, lorsqu'on cherche à obtenir des radiographies nettes d'organes participant aux mouvements respiratoires, tels que le poumon, le rein et la vésicule biliaire.

DER RÖNTGENDOSISZÄHLER MECAPION, SEINE VERLÄSSLICHKEIT UND KONSTANZ*)

von

Ing. Siegmund Strauss, Wien

Zur vollen Beherrschung der Strahlentherapie gehört unbedingt die *Dauermessung*. Mit dem *Mecapion* wird aber nicht nur die Dauer, sondern auch der jeweilige Jetztwert der Strahlung gemessen. Beides geschieht mühelos durch blosse Zeigerablesung ohne jedes Anvisieren. Hiebei wird die jonometrische Methode angewendet, denn nur diese läuft mit den biologischen Wirkungen weitgehend parallel. Auf die Luftionisation durch Röntgenstrahlen gründet sich bekanntlich die deutsche, neuestens auch die amerikanische Röntgendosiseinheit und beide sind nunmehr in dem internationalen »r» vereinheitlicht.

1. Das Prinzip

Die von den Strahlen getroffene Fingerhutkammer leitet von einem geladenen Kondensator eine ganz bestimmte Elektrizitätsmenge ab. Ist diese abgeflossen, so hat eine genau definierte Röntgendosis die Jonenkammer passiert und somit die behandelte Körperstelle getroffen. Die beendete Entladung dieses Kondensators zeigt das *Mecapion* selbsttätig an, indem ein Zeiger auf einem Ziffernblatte sich um einen Skalenteil weiterbewegt. Der Kondensator des Mecapions wird nun *immer wieder u. zw. durchaus selbsttätig* auf ein ganz bestimmtes Potential aufgeladen, worauf ihn die Strahlung immer wieder bis zu einem ganz bestimmten unteren Potential entlädt. Jeder einzelne dieser periodischen Vorgänge wird angezeigt. Die Zahl der Sprünge, die der Zeiger der Messuhr während der ganzen Bestrahlung macht, ergibt die gesamte verabreichte Dosis, die einfach und *ohne jede Umrechnung* (Nomogramm u. dgl.) abgelesen wird.

Der durchaus selbsttätige Ladevorgang des Messkondensators wird durch eine kleine Elektronenröhre in eigenartiger, patentgeschützter Weise, wie unten näher beschrieben, herbeigeführt. Die Röhre hat eine besondere Bauart (selbständig angeordnetes Gitter mit gesonderter

*) Nach einem auf dem II. Internationalen Radiologenkongress in Stockholm gehaltenen Vortrag.

Herausführung) und ist aus hochisolierendem Glas hergestellt. Bei ihr wird im allgemeinen nicht die Verstärker- sondern nur die *Ventilwirkung* ausgenützt, die darin besteht, dass bei entsprechend negativ geladenem Gitter kein Anodenstrom in der Röhre fliessen kann. Das hat den wichtigen Vorteil, dass es auf die genaue Einhaltung der Heizung und der Anodenspannung nicht ankommt.

Solange der Kondensator, der mit dem Gitter unmittelbar verbunden ist, noch eine bestimmte negative Ladung aufweist, kann also ein Anodenstrom in der Röhre nicht auftreten. Erst nach Abfliessen der Ladung bewirkt der plötzlich einsetzende Anodenstrom durch Betätigung eines Relais die neuerliche Aufladung aus einem eigenen kleinen Transformator.

Da mit dem Mecapion grundsätzlich auch Hochohmwiderstände (in *Me*gohmen) *Ca*pacitäten und *Ion*isationen gemessen werden können, ergab sich die Benennung Mecapion dieses Gerätes.

2. Beschreibung der Schaltvorgänge und der Einrichtung des Mecapions

Abbildung 1 zeigt das Schema des Mecapions in der neuesten, verbesserten Schaltung. Die Verstärkerröhre V, deren Glühkathode durch die Batterie B 1 geheizt wird, liegt mit ihrer Anode und dem Anodenrelais R 1 im Stromkreis der Anodenbatterie B 2. Beim Einschalten der Heizbatterie B, wird der Anker dieses Relais R 1 angezogen, und der Kontakt bei 1, 2 geschlossen. Die Batterie B 1 bringt die Klingel K zum Tönen und das rote Schaulämpchen S 1 zum Aufleuchten, während der Elektromagnet R 2 der Schaltuhr U seinen Anker betätigt. Aber erst am Ende der Bewegung dieses Ankers wird der Trennkontakt T geöffnet. Dies hat zunächst zur Folge, dass ein schwacher, aus der Batterie B 1 über die Primäre P und den Vorschaltwiderstand W fliessender Strom plötzlich unterbrochen wird. Dadurch entsteht in der Sekundären S des Transformators Tr ein Spannungsstoss, welcher Folgendes bewirkt:

Zunächst ladet er den durch einander nahe und parallel geführte Leitungen L 1 und L 2 gebildeten Kondensator (strichliert angedeutet), ferner die Jonenkammer D und das Gitter G der Verstärkerröhre V auf, u. zw. auf einen (tieferstehend näher beschriebenen) stark negativen Wert, so dass nunmehr der Anodenstrom in der Verstärkerröhre V vollkommen unterbunden ist. Der Anker des Anodenrelais R 1 fällt daher ab, der Kontakt 1, 2 des Lokalstromkreises wird wieder unterbrochen, weshalb die Klingel aufhört zu tönen, das Lämpchen S 1 zu leuchten aufhört und der Anker der Schaltuhr, sowie auch der Trennkontakt T in ihre Ruhelagen zurückkehren. Zwischen dem Schaltvorgang des Anziehens des Anodenrelais R 1 und der Oeffnung des Trennkontaktes T liegt nur ein kurzer Zeitraum (0.05 Sek.), der nämlich zur Betätigung

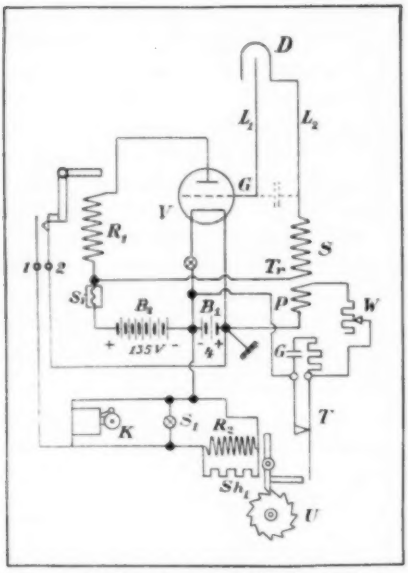


Fig. 1. Verbessertes Schaltbild des Röntgen-Mecapions.

des Ankers der Schaltuhr und dessen Abfallen benötigt wird, so dass die Klingel K nur sehr kurzzeitig tönen kann und das Lämpchen S 1 lediglich aufblitzt. Der Zeiger der Uhr U hat gleichzeitig auf der Skalenscheibe einen Sprung ausgeführt, u. zw. nach rückwärts, wie später noch erläutert wird. Durch die stark negative Aufladung des Gitters der Verstärkerröhre ist die Elektrizitätsmenge, die sich auf den Kondensator-Zuleitungen L 1 und L 2, sowie auf der Jonenkammer D und dem Gitter D angesammelt hat, nunmehr festgehalten. Erst wenn die Jonenkammer von Röntgenstrahlen getroffen wird, entlädt sich der Kondensator allmählich, u. zw. bis zu jenem Punkte, bei welchem der Anodenstrom in der Verstärkerröhre V wieder einzusetzen vermag. Der Anker des Anodenrelais R wird also wieder angezogen, der Trennkontrakt T geöffnet, der Verriegelungs-Spannungsstoss erfolgt abermals und das Spiel beginnt von neuem.

Während nun bei den früheren Ausführungen des Mecapions die Sekundäre mit der Primären des Transformators (als Autotransformator) verbunden war, hat es sich inzwischen als zweckmässig erwiesen, die Sekundärwicklung zu trennen und sie an den *positiven* Pol der Anoden-

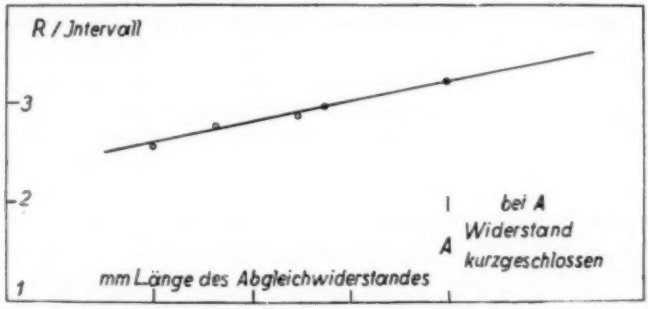


Fig. 2. Lineare Abhängigkeit der R-Dosis pro Signalintervall gemäss dem Vorschaltwiderstande im Primärkreise des Verrieglungstransformators.

batterie zu legen. Das hat den wesentlichen Vorteil, dass sowohl der untere, wie auch der obere Spannungswert, der an der Jonenkammer D wirksam ist, sich immer um die Spannung der Anodenbatterie erhöhen muss. Solcherart beträgt also selbst der untere Spannungswert noch immer 150 Volt, so dass auch bei den stärksten Strahlungen, die in der Tiefentherapie angewendet werden, die Jonenkammer D *voll gesättigt* und dadurch die *Linearität der Mecapionmessangaben gesichert* ist.

Der Vorschaltwiderstand W hat die wichtige Aufgabe, dass die Spannung, welche an der Kammer und am Gitter wirksam ist, wählbar eingestellt werden kann. Abbildung 2 zeigt den vollkommen linearen Verlauf dieser sogenannten Verriegelungsspannungen gemäss der Millimeterteilung des Widerstandes. Auf diese überaus einfache Weise ist es möglich, schon bei der Eichung des Mecapions einen solchen Widerstandswert, u. zw. ein für allemal einzustellen, dass *ein Signal-Intervall* eine abgerundete »r»-Zahl aufweist, in den meisten Fällen *3.0* »r», in welchem Masstabe auch bereits das Ziffernblatt der Schaltuhr U eingeteilt ist. Der Zeiger zeigt unmittelbar die Dosis in »r» für alle Therapiespannungen. Somit werden umständliche und nicht selten zu Irrtümern Anlass gebende Umrechnungen von vornherein vermieden.

Unter der Einwirkung der Röntgenstrahlung auf die Kammer D »läuft» nun das Mecapion, bezw. dessen Zeiger ab, u. zw. wird dies um so rascher geschehen, je stärker die Strahlung ist. Vorher, also bei Beginn der Bestrahlung, wurde dieser Zeiger auf dem Zifferblatte auf jene Röntgendosis eingestellt, der dem Patienten verabreicht werden soll. Sobald der Zeiger die Nullstellung erreicht hat, tritt nun ein im Schema nicht gezeichneter Kontakt in Tätigkeit, welcher den Trennkontakt T kurzschliesst. Infolgedessen bleibt der Anodenstrom in der Verstärkerröhre V weiter bestehen, also auch der Anker des Anodenrelais R 1 angezogen, weshalb das Lämpchen S 1 dauernd leuchtet und die Klingel K fortdauernd tönt, so dass die Bedienungsperson unbedingt auf die Beendigung der Röntgenstrahlung aufmerksam gemacht wird und das Mecapion bezw. die Röntgenapparatur abschaltet.

Damit der Trennkontakt T immer und zuverlässig in durchaus gleichartiger Weise arbeitet, von dessen Funktion ja die Verriegelungsspannung am Gitter der Verstärkerröhre abhängt, liegt parallel zu diesem Kontakte ein Kondensator C geeigneter Grösse mit hintereinander geschaltetem ausgewähltem Widerstand, wodurch dieser Vorgang nunmehr rein elektrisch gestaltet und daher von den mechanischen Eigenschaften der Federn dieses Trennkontaktes, wenn sie erst einmal richtig eingestellt sind, weitgehend unabhängig gemacht ist. Das Aeussere des Apparates zeigt Abbildung 3.

Die Stromquellen, also die 4 Volt-Heizbatterie, sowie die Anodentrockenbatterie von 135 Volt befinden sich im untern Teil des Apparat-

Fig. 3. Ansicht des Röntgen-Mecapions. $\frac{1}{2\,5}$ nat. Grösse.

kastens; der positive Pol der Heizbatterie ist geerdet. Der Apparatkasten läuft auf grossen Gummirollen in Kugellagern, ist daher leicht verschiebbar. Im oberen Teile des Kastens sind die Schaltuhr mit dem Anodenrelais, das Schaulämpchen S_1, die Klingel K und das Vorschaltlämpchen S 2 untergebracht. Von den zwei Buchsenpaaren auf der Schaltplatte wird noch später die Rede sein. Dadurch, dass das Lämpchen S 2 mit dem Faden der Verstärkerröhre hintereinander geschaltet ist, würde das Nichtaufleuchten bei eingeschaltetem Mecapion das Durchbrennen des Heizfadens dieser Röhre anzeigen, ein Fall, der sich allerdings bisher im praktischen Betriebe noch nicht ereignet hat. Auf der Rückseite des Mecapions ist in einem eigenen Schalt-Gusskasten ein 4 m langer Metallschlauch befestigt, welcher die vier Zuleitungen trägt u. zw. eine Heizleitung (die andere liegt an Körper), eine Zuleitung zur Primärwicklung des Transformators, eine Anodenleitung und eine besondere Leitung zur konstanten Erhöhung des Kammerpotentials, wie früher besprochen.

Am andern Ende dieses Kabels befindet sich eisen- und bleigeschützt das Röhrengehäuse, in welchem der Transformator und die Verstärkerröhre V untergebracht sind. An das Röhrengehäuse schliesst sich ein enges, gleichfalls eisen- und bleigeschütztes Rohr an, welches vorne die Jonenkammer trägt. Eine Schutzhülse bewahrt diese Kammer vor Bruch.

3. Unterschied zwischen einem Dosismesser und einem Dosiszähler

Es ist wohl angebracht, auf diese Unterschiede ausdrücklich hinzuweisen. Ein Elektrometer in den verschiedenen Abarten, wie sie zur Ermittlung des Jetztwertes von Röntgenstrahlendosis meist verwendet werden, stellt einen Dosis*messer* dar. Das Elektrometer wird meist mit Hilfe einer kleinen Elektrisiermaschine auf einen ganz bestimmten Spannungswert aufgeladen und durch Anvisieren des Fadens des Elektrometers mittels eines Okulars oder eines Mikroskops oder unmittelbar auf einer beleuchteten Skala wird die Zeit möglichst sorgfältig und genau abgestoppt, die der Faden des Elektrometers benötigt, um unter dem Einfluss der Röntgenstrahlung von einer ganz bestimmten oberen Einstellung

der Spannung zu einer ganz bestimmten unteren zu gelangen. Gleichartig kann nun auch beim Mecapion dieser Zeitablauf gemessen werden. Es besteht aber hier der grosse Unterschied, dass beim Mecapion diese Messung ungleich einfacher von statten geht. Denn sowohl die stete Einhaltung des oberen Spannungswertes, als auch die des unteren Spannungswertes, bei welcher nämlich das Anodenrelais wieder anspricht, ist durch die Eigenart der Mecapion-Schaltung ein für allemal gewährleistet. Da beide Spannungen beim Mecapion von vornherein gegeben sind, müssen sie also nicht erst, wie beim Elektrometer, mit grosser Aufmerksamkeit aufgesucht bezw. ausgewertet werden. Das Intervall zwischen zwei Zeigersprüngen ist allein für die Messung mit dem Mecapion massgebend. Besonders in Verbindung mit der tieferstehend beschriebenen Elektro-Stoppuhr entfällt jede mühsam durch den Beobachter auszuführende Messung, das Anvisieren eines Quarzfadens u. dgl. mit dem koinzidierenden Abstoppen der Uhr, die oft unbequeme, daher ermüdende Haltung des Beobachters, die grosse Inanspruchnahme seiner Aufmerksamkeit u. s. f. Die Messungen erfolgen also durchaus selbsttätig, wodurch sie in der denkbar vollkommensten Weise vereinfacht sind. Ausserdem werden sie aber auch noch genauer, denn bei Benützung dieser vervollkommneten Stoppuhr entfällt überhaupt jeder persönliche Beobachtungsfehler.

Zum Unterschied von einem Intensitätsmesser wirkt aber das Mecapion vor allem auch als *selbsttätiger Zähler*, d. h. die oben besprochenen Zeitintervalle, welche für den Jetztwert der Röntgenstrahlung massgebend sind, werden durch das Gerät selbst addiert (integriert). Alle Faktoren, welche die Ausbeute an Röntgenstrahlen beeinflussen können, werden automatisch mitgezählt, sobald nur die Fingerhutkammer an die richtige Stelle des Bestrahlungsfeldes gebracht und das Mecapion eingeschaltet ist.

4. Die Einstellung der zu verabreichenden Dosis

Diese geschieht in einfachster Weise dadurch, dass vor dem Beginn der Bestrahlung der Zeiger des Mecapions auf eine bestimmte Zahl auf dem Zifferblatte je nach der gewünschten Dosis gebracht wird. Läuft der Zeiger während des darauffolgenden Röntgenbetriebes ab und wird damit das Ende der zu verabfolgenden Dosis erreicht, so ertönt ein andauerndes und schrilles Glockensignal, ferner leuchtet die rote Warnungslampe dauernd auf. Beides geschieht so lange, bis das Mecapion abgeschaltet wird. Diese Massnahmen erinnern also auf das eindringlichste an das Abschalten der Röntgen-Apparatur. Die Ausschaltung kann übrigens auch durch ein eigenes Relais selbsttätig erfolgen.

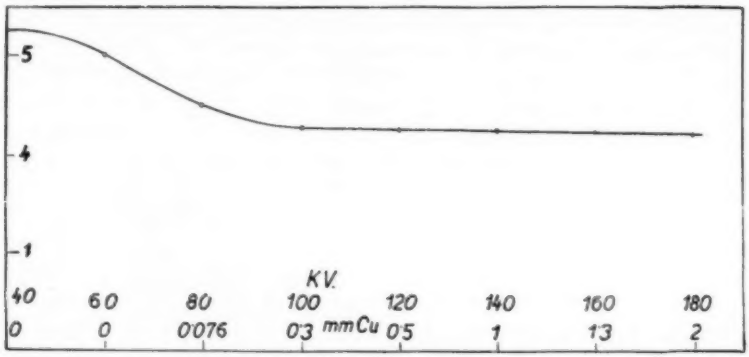


Fig. 4. Beziehung der Halbwertschichten zu dem R/Intervall. Ab 90 KV ergibt sich praktisch Wellenlängenunabhängigkeit der Ionenkammer.

5. Messungen der Strahlenintensität

Misst man mit dem Mecapion die Zeit, die zwischen zwei auf einanderfolgenden Zeigersprüngen bei einer bestimmten Bestrahlung vergeht, so gibt dieses Zeitintervall ein unmittelbares Mass der jeweiligen Strahlenintensität. Diese Zeitmessung kann mit jeder Stoppuhr erfolgen. Wie schon oben erwähnt, kann dies aber auch mit einer eigens dazu gebauten automatisch arbeitenden Stoppuhr geschehen, deren Elektromagnet selbsttätig die Uhr ein- und ausschaltet. Zu diesem Zwecke wird die Stoppuhr an das eine auf der Schaltplatte befindliche Buchsenpaar angesteckt.

6. Linearität (Proportionalität)

Die Angaben des Mecapions sind durchaus linear, d. h. die Beziehung von Röntgendosis pro sek. zu den Signalintervallen, in einem Koordinadensystem aufgetragen, verläuft in lauter *Geraden*. Die doppelte Strahlenmenge ergibt also halb so lange Signalintervalle als die ursprüngliche u. s. f.

7. Der Vorgang der Messung

Die Schutzhülse der Jonenkammer wird entfernt und diese selbst an die Körperstelle gebracht, die bestrahlt werden soll. Wird mit Presstubus gearbeitet, so wird dieser seitlich, u. zw. möglichst nahe am Pressrande angebohrt (23 mm ϕ) und in die Oeffnung das Kammerrohr eingeführt. Hierauf wird der Knebel am Mecapion in die Einschaltstellung gedreht, wobei die grüne Lampe dauernd aufleuchten muss, die rote dagegen,

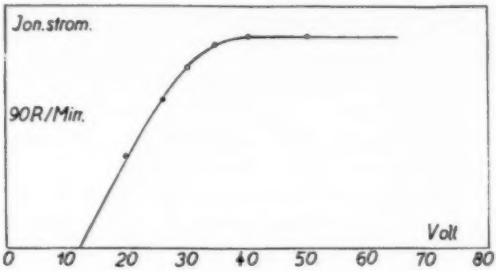


Fig. 5. Sättigung der Mecapionkammer selbst für die starke Strahlung von 90 R/min schon bei 40 Volt, während die Mindestspannung an der Kammer 150 Volt beträgt.

wenn das Mecapion längere Zeit nicht im Betriebe war, nur aufblitzt. Zugleich ertönt ein kurzzeitiges Glockensignal. Hierauf wird die zu verabreichende Dosis, mit dem Zeiger an der Uhrskala eingestellt. Mit der Bestrahlung kann nun unmittelbar begonnen werden. Während derselben folgen Licht- und Glockensignale aufeinander, an deren Rhythmus sich die Bedienungsperson erfahrungsgemäss rasch gewöhnt und welche in der denkbar bequemsten Weise den Ablauf der Dosis fortdauernd zu kontrollieren gestatten. Dabei geht der Zeiger immer um einen Skalenteil zurück, bis er die Nullstellung erreicht hat. Ein lautes und andauerndes Glockensignal kündigt hierauf den Ablauf der Dosis an. Die Schnelligkeit des Ablaufes richtet sich ganz nach der Stärke der Bestrahlung (abhängig von der Röntgenapparatur, -Röhre, Spannung, Filter, Abstand, Feldgrösse) und entspricht ihr durchaus *selbsttätig*.

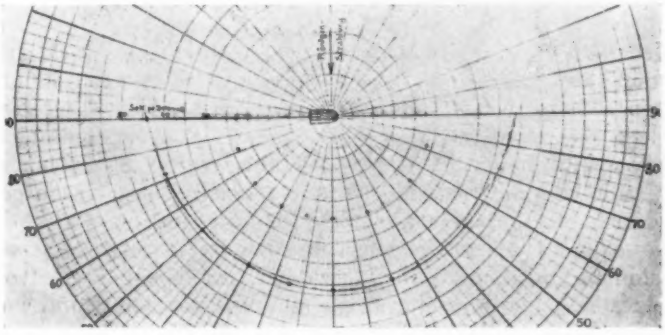


Fig. 6. Richtungsunabhängigkeit der Mecapionkammer.

8. Sättigung und Wellenlängenunabhängigkeit der Mecapion-Kammer

Nach Messungen der Physikalisch-Technischen Reichsanstalt, Berlin-Charlottenburg, und anderer befugter Eichstellen an der Fingerhutkammer des Mecapions ergab sich selbst bei Strahlungen von 90 R pro min., wie sie in der Tiefentherapie-Praxis kaum auftreten, noch immer eine vollständige Sättigung der Jonenkammer. Wie die Abb. 5 zeigt, ist die Kammer schon bei 40 Volt gesättigt, während, wie wir oben gesehen haben, der unterste Spannungswert an der Kammer sogar 150 Volt beträgt. Es sei darauf hingewiesen, dass ferner die Kammer praktisch auch *ohne jede Wellenlängenabhängigkeit* ist, was aus der Abb. 4 hervorgeht, welche die Halbwertschichten in Bezug zu den R pro Signalintervall zeigt und somit dartut, dass von 90 K V angefangen bis zu den höchsten Spannungen praktisch so gut wie keine Härteabhängigkeit der Kammer besteht.

9. Richtungsunabhängigkeit der Mecapion-Kammer

Gleichfalls nach Messungen der Physikalisch-Technischen Reichsanstalt ergab sich eine fast vollständige Richtungsunabhängigkeit der Fingerhutkammer des Mecapions lt. Abb. 6. Es ist daher durchaus nicht nötig, dass die Kammer senkrecht von den Strahlen getroffen wird, was die Arbeiten am Patienten mitunter nicht unwesentlich erleichtert. Das gleiche gilt natürlich bei Messungen im Wasserphantom (siehe Punkt 12 f). Hiezu kommt, dass die Versuche, auch *die Zuführung zur Jonenkammer röntgenschattenfrei* zu gestalten, bereits günstig verlaufen sind und unmittelbar vor ihrem Abschlusse stehen, so dass auch nach dieser Hinsicht die neuesten Fortschritte beim Mecapion bereits Anwendung finden.

10. Die Selbstkontrolle

Das Mecapion kontrolliert, was kein anderes Dosismessgerät zu leisten vermag, die Bestrahlung und seine Wirkungsweise weitgehendst und vollkommen selbsttätig. Wird z. B. ein zu schwaches Filter eingelegt, versehentlich fortgelassen, oder sonst irgend eine Einstellung an der Röntgenapparatur nicht richtig durchgeführt, so ist die Glockensignalfolge eine andere. Das Ohr, das sich an den Rhythmus rasch gewöhnt, bemerkt dann sofort, dass etwas nicht in Ordnung ist.

Sollte aber am Dosiszähler selbst ein Defekt auftreten, beispielsweise ein Verklemmen und damit Stillstehen der Uhr, oder auch ein Isolationsfehler (beides ist allerdings durch die überaus sorgsame und gewissenhafte Konstruktion und Durchbildung des Mecapions so gut wie ausgeschlossen), so ertönt ein *Dauerglockensignal* und das Aufleuchten der

roten Lampe hält so lange an, bis das Mecapion abgeschaltet wird. Eine Ueberdosierung ist also selbst aus solchen Anlässen nach menschlichem Ermessen nicht möglich.

11. Selbstablauf (Dunkeleffekt)

Jeder Jonisationsmesser bezw. jedes Elektrometer weist einen sogenannten Dunkeleffekt auf, d. h. auch wenn keinerlei Röntgenstrahlung auf die Kammer wirkt, tritt eine geringe Selbstentladung auf, denn die Isolation erreicht niemals den Wert Unendlich. Immerhin ist es beim Mecapion durch die höchst getriebene Isolation des Gitterpoles, bezw. des Innenpoles der Jonenkammer und durch eigenartige gleichfalls unter Patentschutz stehende Massnahmen gelungen, den Dunkeleffekt so weit herabzumindern, dass die Genauigkeit der Messung überhaupt nicht leidet. Das Messergebnis kann also dadurch praktisch nicht gedrückt werden und jede Korrektur der gemessenen Intervalle entfällt.

12. Zusatzapparate

a) Elektrostoppuhr

Zu Eichzwecken, ferner zur Messung der Intensitäten, Tiefenquotienten, Halbwertschichten u. dgl. müssen die Schlagintervalle selbst genau gemessen werden. Dies geschieht mit jeder Stoppuhr. Zur Erleichterung der Messung und zur Vermeidung der persönlichen Beobachtungsfehler wurde, wie oben angeführt, eine besondere, elektromagnetisch gesteuerte

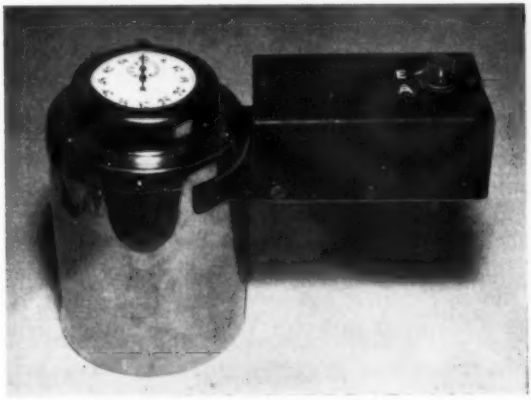


Fig. 7. Elektromagnetisch durch die Signalfolge des Mecapions, also selbsttätig gesteuerte Stoppuhr, welche alle Messungen ideal vereinfacht. Rund 1:4 nat. Grösse.

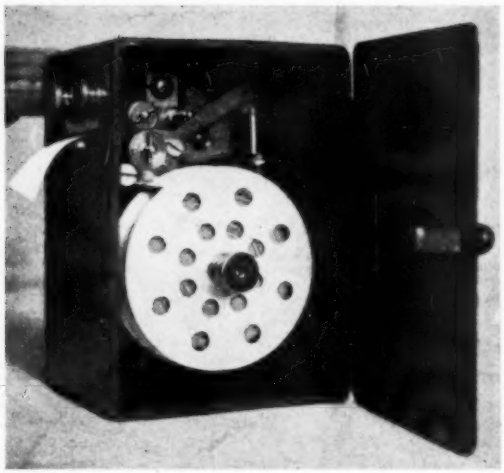


Fig. 8. Dosisnachweiser. Die Länge des beim Arbeiten des Mecapions vorwärts geschobenen (gummierten Morse-) Papierstreifens ergibt unmittelbar die verabreichte Dosis (3 R = 1 mm.) Rund 1/3 nat. Grösse.

Stoppuhr Abb. 7 durchgebildet, die bei Beginn des zu messenden Intervalles selbsttätig in Gang kommt und sich nach dessen Beendigung wieder selbsttätig abstoppt. Auf diese ideale Weise wird das jeweilige Intervall mühelos und ohne jedes Anvisieren vom Ziffernblatt abgelesen.

b) Registriervorrichtung für dokumentarische Kontrolle, der Dosis-Nachweiser

Nicht selten wird eine bleibende Aufzeichnung (dokumentarische Festhaltung) der Dosis erwünscht sein, um ein Minimum an Verantwortlichkeit herzustellen. Hiefür wurde ein spezieller Apparat Abb. 8 konstruiert, welcher an das andere Buchsenpaar gesteckt wird und, wie folgt, in einfacher und betriebssicherer Weise arbeitet: Ein gummierter Papier-Morse-Streifen wird bei jedem Intervall, das 3 »r» entspricht, um einen Millimeter vorwärts geschoben. Nach beendeter Bestrahlung wird der Streifen mittels einer beigegebenen Vorrichtung abgeschnitten. *Seine Länge gibt* also *unmittelbar die Grösse der verabreichten Dosis an.* In die Krankengeschichte eingeklebt und von allen Beteiligten paraphiert, bildet dieser Streifen solcherart ein Dokument, von dem z. B. bei gerichtlichen Fällen mit grossem Vorteil Gebrauch gemacht wird.

c) Stromnormal

Um die Konstanz des Mecapions jederzeit nachprüfen zu können, wurde ein *Stromnormal* Abb. 9 nach BEHNKEN durchgebildet, welches

Fig. 9. Stromnormale (α Strahler = Uranoxyd) zur Nachprüfung der Konstanz des Mecapions. $^1/_5$ nat. Grösse.

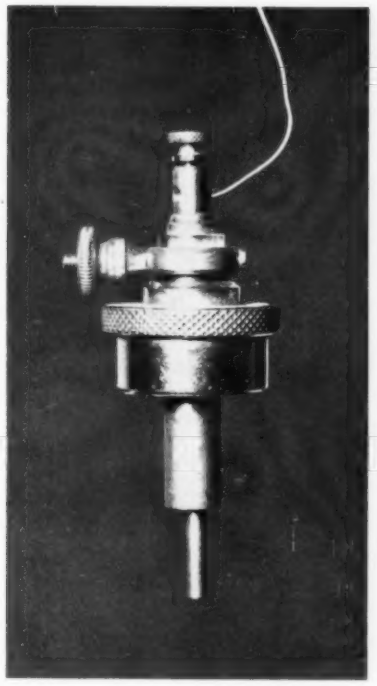


Fig. 10. Elektrodenanschlussklemme zum Mecapion. Mittels derselben können Isolationen (Pertinaxrohre, Hartgummi, Transformatorenöle etc. überprüft werden: in Verbindung mit einer lichtelektr. Zelle auch die UV (ultraviolett-)Strahlung, natürl. u. künstl. Lichtquellen aller Art. Masstab. 1 : 1.

an dem Tubus des Röhrengehäuses anschraubbar ist. Das Stromnormale besteht aus zwei zylindrischen Metallröhren, die mit Uranoxyd bedeckt sind und durch ihre α-Strahlung den Luftzwischenraum zwischen den beiden Belegungen ionisieren. Das Mecapion nimmt nun bei angeschaltetem Stromnormal die Ablaufzeiten desselben auf — bei Benützung der Elektro-Stoppuhr vollkommen selbsttätig — und diese Entladezeit muss bei jeder etwa allmonatlich vorzunehmenden Nachkontrolle unverändert bleiben. Dadurch ist die dauernde Konstanz des Mecapions sichergestellt, bezw. jederzeit kontrollierbar.

d) Elektrodenanschlussklemme

Für sämtliche Messungen, bei denen die Herausführung der beiden Jonenkammerelektroden erwünscht ist, kann an den Tubus des Röhrengehäuses ein besonderes Elektrodenanschluss-Klemmenpaar angeschraubt werden. Abb. 10. Dieses ist natürlich mit einwandfrei guter Bernsteinisolation ausgestattet. Mittels dieser Klemme können nun mit dem Mecapion eine Reihe von Sonder-Messungen vorgenommen werden, beispielsweise die Prüfung der Isolation von Hochspannungs-Transformatoröl, von Porzellan, Pertinax oder Hartgummiröhren etc. Es kann aber auch an diese Spezialklemme eine lichtelektrische Zelle, beispielsweise mit Kadmiumbelag, angeschlossen werden, in welchem Falle das Mecapion als Lichtdosismesser und-Zähler für ultraviolette Strahlen der Sonne, des Himmelslichtes oder auch künstlicher Lichtquellen (Quecksilberdampflampe, Bogenlampe) wirkt. Da das Mecapion den Anschluss verschiedener Hilfsgeräte gestattet, kann auch ein Chronograph, wie er für meteorologische Zwecke vielfach in Verwendung ist, benützt werden, um die Signalintervalle graphisch festzuhalten.

e) Stativ

Zur Befestigung und zur leichten und bequemen Einstellung, bezw. Handhabung des Röhrenkammerteiles dient ein Stativ mit Spezial-Kreuzkopfklemme. Letztere ist so eingerichtet, dass die Jonenkammer mit einem einzigen Handgriff in jede Stellung gebracht werden kann.

f) Wasserphantom

Die Jonenkammer des Mecapions kann, gleichwie bei anderen Geräten mit Fingerhutkammern, in Körperhöhlen eingeführt werden. Die Bestimmung der prozentuellen Tiefendosis erfolgt mit einem Wasser-

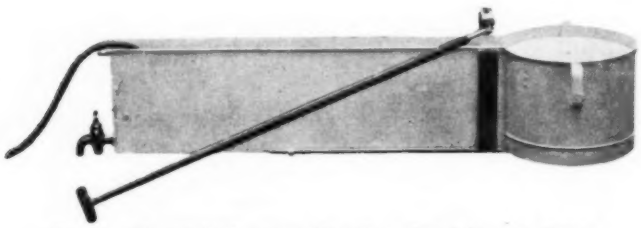


Fig. 11. Wasserphantom zum Mecapion. Rund 1/14 nat. Grösse.

phantom lt. Abb. 11 in der üblichen Weise. Ein beigegebener entsprechend geformter Gummischlauch wird über die Kammer und die beiden Rohre gestülpt, wodurch jede besondere Abdichtung der Kammer entfällt.

ZUSAMMENFASSUNG

1. Exakte Dosierung der Bestrahlung nach »r»-Einheiten und selbsttätige Kontrolle.
2. Selbsttätige Kontrolle der richtigen Filtereinlage durch den Rhythmus der Glockensignale.
3. Selbstanzeige der beendeten Dosis, sowie ev. Störquellen.
4. Objektive und einfachste Messung der jeweiligen Strahlenintensitäten.
5. Bestimmung der Halbwertschicht und damit auch der effektiven Wellenlänge und anderer wichtiger Strahlungsfaktoren.
6. Denkbar einfachste Bedienung und Handhabung des Gerätes.
7. Stabile Ausführung bereits im strengsten Betrieb dauernd bewährt (kein blosses Laboratoriumsgerät).
8. Vielseitige andere Anwendungsmöglichkeiten.

Das Mecapion hat sich vermöge seiner besonderen und durchaus eigenartigen Vorzüge schon zahlreiche Anhänger erworben. Es hält jeder Kritik stand und erfüllt die Bedürfnisse der Röntgenpraxis in vollendeter Weise.

SUMMARY

1. Exact dosage of radiation in »r»-units and automatic control.
2. Automatic controll of the right filter put in by the rhythm of the clock signal.
3. Automatic warning of completed dose as well as possible sources of error.
4. Objective and simple measurement of the present intensity of radiation.
5. Estimation of the semi-value thickness and thereby also the effective wave-length and other important factors pertaining to the radiation.
6. The simplest possible utilization and handling of the instrument.
7. The stable execution has already stood the test under exacting running conditions (not only laboratory instrument).
8. A great many other useful possibilities.

The mecapion has on account of its particular and throughout peculiar advantages already attracted a great many advocates. It holds its own against every criticism and fills the need in röntgenological practice in a perfect way.

RÉSUMÉ

1. Dosage exact de l'irradiation en unités »r» et contrôle automatique.
2. Contrôle automatique du filtre convenable par le rythme de la sonnerie signalisatrice.
3. Indication automatique de la dose terminale et des sources éventuelles de perturbation.
4. Mesure objective et simple de toutes les intensités de rayons.
5. Détermination de la couche de demi-valeur et par suite de la longueur d'onde effective ainsi que des autres facteurs importants d'irradiation.

6. Maniement et fonctionnement des plus simples de l'appareil.
7. Construction solide, à l'épreuve de l'usage le plus intensif (rien de commun avec un appareil de laboratoire..
8. Possibilité d'usages multiples).

Par ses qualités remarquables et particulières, le Mecapion a déjà acquis des nombreux partisans. Il résiste à toute critique et répond dans la mesure la plus étendue aux exigences de la pratique radiologique.

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REPORT ON CASES RADIOLOGICALLY TREATED AT RADIUMHEMMET IN STOCKHOLM 1921—1927 AND CONTROLLED 1921—1927

BY

ELIS BERVEN AND JAMES HEYMAN
GENERAL DEPARTMENT. GYNECOLOGICAL DEPARTMENT.

35—280376. *Acta Radiologica. Vol. IX. 1928.*

INTRODUCTION.

The following report comprises all cases treated at Radiumhemmet during 1921—1927. Patients having been treated before 1921 are not reported in this statistics, except the cases of carcinoma of the uterus.

We want our statistics to illustrate in the first place the number of patients which after treatment are free from subjective and objective symptoms out of the total number of applicants, as well as the permanency of the healing.

In the second place we want to inform about the palliative effect of the therapy i. e. the objective improvement of the symptoms and subjective relief of the troubles in all cases where full freedom from symptoms are not obtained as well as about the duration of this palliative effect.

The statistics therefore have been arranged in following groups:

I. Symptomfree.

II. Symptomfree. Dead from intercurrent disease.

III. Symptomfree, then recurrence. Retreated and later symptomfree or improved.

IV. Symptomfree, then recurrence. Retreated and later progression. Hopeless.

V. Improved.

VI. Temporary improved. Then progression or dead.

Table I reports all cases radiologically treated 1921—1927.

Table II reports cases treated with electro-endothermy and desiccation 1927.

Table III reports all cases of cancer colli uteri treated 1914—1927 and Table IV all cases of cancer corporis uteri 1913—1927.

Table I. Report on cases radiologically treated at Radiumhemmet

	Diseases	Results of: Symptom-free							Symptom-free. Dead from intercurrent disease							Symptom-free, then recurrence. Retreated and later symptom-free or improved						
		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Blood and Bloodgenerating Organs:																								
1	Polyglobulia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
2	Lymphadenitis subacuta	1	2	4	4	7	9	1	*28*	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	*2*
3	Lymphomata tbc.	28	25	12	22	22	26	12	*147*	—	—	—	—	1	—	—	*1*	8	4	5	2	—	—	—	*19*
4	Morbus Mikulicz	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
5	Leukämia lymphatica	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
6	Myelosis aleucæmia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
7	Lymphogranulomatosis	—	1	1	2	2	1	—	*7*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
8	Actinomycasis colli	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
9	Tumor colli	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
10	Tumor mal. colli non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
11	Tumor mal. colli op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
12	Cancer lymphoglandularum	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
13	Cancer lymphogl. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
14	Cancer lymphogl. op.*)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
15	Lymphosarcoma	—	—	—	2	1	1	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
16	Lymphosarcoma non rad. op.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
17	Lymphosarcoma op. rec.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	1	—	*1*	—	1	—	—	—	—	—	*1*
18	Lymphosarcoma op.*)	—	—	—	—	2	—	—	*2*	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	*2*
19	Hæmangioma vinosum	4	6	—	3	—	—	—	*13*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
20	Hæmangioma cavernosum	15	20	24	28	20	19	2	*128*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
21	Hæmangioma cavernosum op.*)	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
22	Angioblastoma op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
23	Angioblastoma op.*)	—	—	1	—	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
24	Lymphangioma	—	2	1	—	—	1	—	*4*	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*
25	Lymphangioma non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
26	Lymphangioma op, rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
27	Lymphangioma op.*)	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
28	Angiofibroma	—	—	—	2	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
29	Angiosarcoma	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
30	Angiosarcoma non. rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
31	Angiosarcoma op. rec.	—	—	1	—	—	—	1	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
32	Angiosarcoma op.*)	—	1	1	1	—	—	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
33	Endothelioma	—	1	—	—	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
34	Endothelioma op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
35	Endothelioma op.*)	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

in Stockholm 1921—1927 and controlled 1921—1927.

the treatment																																		
Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																																		
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	*2*	—	3	1
19	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	*5*	*7*	*20*	63	2
—	—	—	—	—	—	—	—	—	2	1	4	5	18	39	*69*	—	—	—	—	—	—	—	*4*	*15*	*67*	*101*	423	3
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	2	—	—	—	—	3	4
—	—	—	—	—	—	—	—	—	—	—	—	—	6	*6*	4	—	—	—	—	—	—	4	8	—	4	—	22	5
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1	1	—	—	—	2	6
—	—	—	—	—	—	—	—	—	—	—	—	2	2	*4*	4	2	—	—	—	—	—	6	*12*	*1*	*3*	*2*	35	7
—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	1	8
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	1	9
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	*1*	—	—	1	10
—	—	—	—	—	—	—	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	1	11
—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	2	—	2	1	6	12
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1	2	—	—	—	3	13
1	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	14
2	—	1	—	—	—	*3*	—	—	—	—	—	1	2	*3*	4	1	—	—	—	—	5	*19*	*1*	—	*3*	39	15
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	16
2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	2	8	*1*	*1*	—	15	17
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	18
—	—	—	—	—	—	—	3	1	1	7	—	3	3	*18*	—	—	—	—	—	—	—	3	—	*20*	*109*	163	19
—	—	—	—	—	—	—	4	5	15	19	26	45	47	*161*	1	—	—	—	—	—	1	3	22	*37*	*69*	421	20
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	21
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	1	22
—	—	—	—	—	—	—	—	—	1	—	1	—	1	4	—	—	—	—	—	—	—	—	—	—	—	—	2	23
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	3	—	2	1	15	24
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	*1*	—	2	25
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	1	26
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	27
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	4	28
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	29
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	30
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	31
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	4	32
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	—	1	2	9	33
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	1	—	—	—	—	—	1	—	—	—	—	2	34
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	2	35

	Diseases	Results of Treatment																									
		Symptom-free								Symptom-free. Dead from intercurrent disease								Symptom-free, then recurrence. Retreated and later symptom-free or improved							
		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Nose and Throat:																								
36	Ulcus cavitatis nasi	2	1	—	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
37	Tbc. cavitatis nasi	1	—	—	1	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
38	Tumor benign. cavit. nasi	1	3	—	1	5	—	—	10	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1
39	Tumor benign. cavit. nasi op. rec.	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
40	Tumor malign. cavit. nasi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
41	Cancer cavitatis nasi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
42	Cancer cavit. nasi non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
43	Cancer cavit. nasi op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
44	Cancer cavit. nasi op.*)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
45	Sarcoma cavitatis nasi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
46	Sarcoma cavit. nasi non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
47	Sarcoma cavit. nasi op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
48	Sarcoma cavit. nasi op.*)	—	—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
49	Tumor maxillæ	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—
50	Tumor maxillæ non rad. op.	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
51	Tumor maxillæ op. rec.	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
52	Tumor maxillæ op.*)	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
53	Tumor malign. maxillæ op.*)	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
54	Actinomycosis maxillæ	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
55	Cancer maxillæ inop.	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—
56	Cancer maxillæ non. rad. op.	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
57	Cancer maxillæ op. rec.	—	—	—	1	—	—	1	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
58	Cancer maxillæ præop. treatment op.*)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
59	Cancer maxillæ op.*)	2	3	1	1	—	5	—	12	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
60	Sarcoma maxillæ inop.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	1
61	Sarcoma maxillæ non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
62	Sarcoma maxillæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	1
63	Sarcoma maxillæ op.*)	1	1	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
64	Pansinusitis	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
65	Cancer sinus frontalis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
66	Ethmoiditis	—	—	—	3	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
67	Abscessus retrobulb. post ethmoiditis	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
68	Tumor reg. ethmoidal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
69	Tumor reg. ethmoidal, op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
70	Cancer reg. ethmoidal	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
71	Sarcoma reg. ethmoidal. non rad. op.	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
72	Tbc. nasopharyngis	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
73	Diphteritis nasopharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1
74	Adenoides nasopharyngis	—	—	5	2	2	—	—	9	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
75	Tumor nasopharyngis	2	—	2	1	—	3	—	8	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment

Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																											
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	6	36
—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	—	—	—	—	—	—	—	—	—	1	1	4	37
1	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	3	4	7	28	38
—	—	—	—	—	—	—	—	—	—	—	—	2	1	3	—	—	—	—	—	—	—	—	1	1	4	10	39
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	1	40
—	3	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	6	41
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	42
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	1	43
—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	44
—	—	—	—	—	—	—	—	—	—	—	—	1	1	2	2	—	—	—	—	—	2	3	—	—	—	7	45
1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	46
—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	47
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	48
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	1	1	5	49
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	50
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	51
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	52
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	53
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	54
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	3	—	—	—	—	—	3	16	—	1	14	36	55
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5	—	—	—	6	56
—	2	—	—	—	—	2	—	—	—	—	—	—	1	1	1	—	—	—	—	—	1	6	1	—	9	22	57
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	1	1	2	—	5	58
—	3	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	12	—	3	1	31	59
1	—	—	—	—	—	—	—	—	1	—	—	—	—	1	—	—	1	—	—	—	1	3	—	—	3	9	60
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	1	—	4	61
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	1	—	—	—	3	62
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	5	63
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	1	3	64
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	65
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	4	66
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	67
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	68
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	69
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	70
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	71
1	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	2	72
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	73
—	—	—	—	—	—	—	—	—	—	1	1	1	—	3	—	—	—	—	—	—	—	—	—	7	15	34	74
1	—	—	—	—	—	1	—	—	2	—	—	1	2	5	4	—	—	—	—	—	4	3	—	8	3	32	75

	Diseases	Results of the Symptom-free: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Nose and Throat (continued):																								
76	Tumor nasopharyngis op. rec.	1	—	1	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
77	Tumor malign. nasopharyngis	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
78	Cancer nasopharyngis	1	—	—	1	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
79	Cancer nasopharyngis op. rec.	—	2	—	1	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
80	Cancer nasopharyngis op. *)	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
81	Sarcoma nasopharyngis	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1
82	Sarcoma nasopharyngis non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
83	Rhinitis chron. hyperpl.	—	—	1	1	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
84	Tonsillitis chronica	24	83	84	58	65	54	24	392	—	1	1	—	—	—	—	2	—	—	1	—	—	—	—	1
85	Abscessus post tonsillectomia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
86	Ulcus tonsillæ	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
87	Tbc. tonsillæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
88	Tumor tonsillæ	—	—	1	—	1	5	—	7	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
89	Cancer tonsillæ	—	—	—	—	—	2	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
90	Cancer tonsillæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
91	Cancer tonsillæ op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
92	Sarcoma tonsillæ	—	—	—	1	3	2	2	8	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—
93	Sarcoma tonsillæ non rad. op.	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
94	Sarcoma tonsillæ op. rec.	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
95	Sarcoma tonsillæ op. *)	1	—	—	1	—	—	1	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
96	Pharyngitis chronica	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
97	Tbc. pharyngis	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
98	Tumor pharyngis	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
99	Cancer pharyngis	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
100	Cancer pharyngis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
101	Tumor epipharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
102	Cancer epipharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
103	Cancer oropharyngis	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
104	Cancer oropharyngis op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
105	Sarcoma oropharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
106	Tumor hypopharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
107	Cancer hypopharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
108	Sarcoma hypopharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
109	Cancer laryngopharyngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
110	Cancer laryngopharyngis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
111	Tumor benign. laryngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1
112	Tumor benign. laryngis op. rec.	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
113	Cancer laryngis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
114	Cancer laryngis non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
115	Cancer laryngis op. rec.	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

Symptom-free, then recurrence. Retreated and later progression. Hopeless — Duration of cure							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	2	—	—	1	6	76
—	2	—	1	—	—	3	—	—	—	—	—	—	—	—	3	—	—	—	—	—	3	2	—	—	1	6	77
—	—	—	—	—	—	—	—	—	—	—	—	1	2	3	8	—	—	—	—	—	8	15	—	1	1	33	78
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	4	79
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	80
1	1	—	—	—	—	2	—	—	—	—	—	—	1	1	3	1	—	—	—	—	4	4	—	—	—	13	81
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	1	—	—	—	1	82
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	3	1	2	10	83
1	—	—	—	—	—	—	3	3	4	4	14	17	45	90	1	—	1	—	—	—	2	9	23	77	142	738	84
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	1	3	85
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	86
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	87
1	1	—	—	—	—	2	—	—	—	—	—	1	1	2	1	—	—	—	—	—	1	5	—	1	2	18	88
—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	2	—	1	—	—	—	6	16	1	—	7	36	89
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	2	—	—	—	—	2	90
—	1	—	—	—	—	1	—	—	—	—	—	—	2	2	—	—	—	—	—	—	—	—	—	—	—	1	91
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	9	—	—	2	22	92
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	93
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	94
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	95
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	5	6	96
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	97
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	2	98
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	3	99
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	1	100
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	1	101
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	102
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	103
—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	104
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	105
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	—	—	—	3	4	—	3	—	7	106
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	24	2	4	28	61	107
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	108
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	—	—	—	6	109
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	110
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	111
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	3	112
1	—	—	—	—	—	1	—	—	—	—	—	—	1	1	1	—	—	—	—	—	1	8	—	7	—	17	113
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	114
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	115

	Diseases	Results of the Treatment: Symptom-free: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Nose and Throat (continued):																								
116	Cancer laryngis op. *)	1	—	1	—	1	1	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
117	Sarcoma laryngis op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
118	Tumor tracheæ op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
119	Cancer tracheæ op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
120	Asthma bronchiale	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Lungs:																								
121	Tumor pulmonis	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
122	Actinomycosis pulmonis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Pleura and Mediastinum:																								
123	Actinomycosis pleuræ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
124	Cancer pleuræ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
125	Tumor mediastini	—	—	1	—	—	—	—	*1*	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—
126	Cancer mediastini	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Digestive tract:																								
127	Leucoplaciæ labii	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
128	Leucoplaciæ buccæ et linguæ	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
129	Eczema chron. labii	—	—	—	2	1	1	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
130	Cysta labii	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
131	Ulcus simplex labii	1	3	—	1	2	—	—	*7*	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*
132	Tumor benign. labii	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
133	Tumor benign. labii op. rec.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
134	Cancer labii	10	10	11	14	22	15	15	*97*	5	1	3	3	2	—	—	*14*	—	—	1	—	—	2	—	*3*
135	Cancer labii rec. post radiol. treatment	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
136	Cancer labii op. rec.	—	1	5	3	—	3	3	*15*	—	1	—	—	1	1	—	*3*	—	—	—	—	—	—	—	—
137	Cancer labii op. *)	1	2	2	3	1	—	—	*9*	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—
138	Sarcoma labii	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
139	Fistula dentis	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
140	Tumor benign. buccæ	—	1	—	—	—	—	—	*1*	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—
141	Tbc. buccæ	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
142	Actinomycosis buccæ	—	—	—	1	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
143	Cancer buccæ	—	—	3	—	1	3	3	*10*	1	3	—	—	—	—	—	*4*	—	—	—	—	—	—	—	—
144	Cancer buccæ op. rec.	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
145	Cancer buccæ op. *)	1	2	—	1	—	—	—	*4*	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—
146	Sarcoma buccæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
147	Sarcoma buccæ op. *)	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
148	Parotitis subchron.	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
149	Cysta parotidis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
150	Tbc. gland. parotidis	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
151	Actinomycosis gland. parotidis	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment

Symptom-free, then recurrence. Retreated and later progression. Hopeless — Duration of cure: 1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Temporary improved. Then progression or dead: 1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
2	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	116
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	117
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	118
—	—	—	—	—	—	—	—	1	—	—	—	—	1	1	—	—	—	—	—	—	—	1	—	—	—	1	119
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	2	1	6	120
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	3	—	—	2	7	121
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	2	—	—	—	3	122
1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	123
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	2	124
—	—	—	—	—	—	—	—	—	—	—	1	—	1	2	7	—	—	—	—	—	7	4	—	2	2	19	125
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	126
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	7	127
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	22	25	128
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	5	11	129
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	130
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	18	26	131
3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	19	20	132
2	1	—	—	—	—	3	—	—	—	—	—	1	2	3	2	—	—	—	—	—	2	17	5	5	4	153	133
1	1	—	—	—	—	2	—	—	—	—	—	—	—	—	4	—	—	—	—	—	4	12	2	1	5	44	134
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	10	135
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	2	136
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	137
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	4	138
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	139
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	140
6	1	—	—	—	—	7	—	—	—	—	—	—	2	2	2	—	—	—	—	—	2	15	—	2	10	52	141
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	7	1	—	2	12	142
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	1	—	—	—	6	143
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	144
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	145
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	1	146
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	147
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	4	148
																											149
																											150
																											151

	Diseases	Results of: Symptom-free — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Digestive tract (continued):																								
152	Teratoma parotidis	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
153	Teratoma parotidis op. rec.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
154	Teratoma parotidis op. *)	2	—	—	—	—	2	1	*5*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
155	Tumor parotidis inop.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
156	Tumor parotidis non rad. op.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
157	Tumor parotidis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
158	Tumor parotidis op. *)	—	—	2	4	1	—	1	*8*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
159	Cancer parotidis	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
160	Cancer parotidis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
161	Cancer parotidis op. *)	—	—	1	1	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
162	Sarcoma parotidis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
163	Sarcoma parotidis op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*
164	Tumor region. submax.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
165	Cancer region. submax. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
166	Glossitis linguæ	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
167	Ulcus linguæ	—	—	—	1	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
168	Tbc. linguæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
169	Papilloma linguæ	—	—	—	—	2	1	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
170	Tumor linguæ	—	—	—	1	2	—	1	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
171	Tumor linguæ op. *)	—	—	—	1	—	—	1	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
172	Actinomycosis linguæ	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
173	Cancer linguæ	1	—	3	2	2	5	—	*13*	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—
174	Cancer linguæ op. rec.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
175	Cancer linguæ præop. treatment op. *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
176	Cancer linguæ op. *)	2	—	1	—	1	—	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
177	Sarcoma linguæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
178	Sarcoma linguæ non rad. op.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
179	Tumor region. sublingualis	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
180	Cancer region. sublingualis	2	—	—	—	—	3	—	*5*	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	*2*
181	Cancer region. sublingualis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
182	Cancer region. sublingualis op. *)	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
183	Tumor gingivæ	2	1	—	1	—	3	2	*9*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
184	Tumor gingivæ op. *)	1	—	—	1	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
185	Cancer gingivæ	1	1	1	1	1	2	—	*7*	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—
186	Cancer gingivæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
187	Cancer gingivæ præop. treatment op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
188	Cancer gingivæ op. *)	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
189	Sarcoma gingivæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
190	Tumor palat. duræ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
191	Cancer palat. duræ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment

Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																											
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	1	7	152
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	2	—	1	1	3	153
1	—	—	—	—	—	1	—	—	—	—	—	1	1	2	1	—	—	—	—	—	—	1	—	1	1	7	154
—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	—	—	—	—	—	—	—	1	—	—	6	14	155
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	1	2	—	—	5	156
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	4	6	157
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	—	11	158
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	1	4	159
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	2	160
1	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	3	161
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	162
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	163
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	1	2	164
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	165
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	1	2	166
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	16	19	167
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	1	1	4	168
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	26	29	169
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	10	15	170
2	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	171
—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	—	—	—	—	—	—	—	—	—	—	—	1	172
1	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	3	1	22	71	173
—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	27	—	9	6	16	174
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	3	175
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	5	176
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	2	177
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	178
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	179
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	3	—	1	4	27	180
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	12	—	—	—	1	181
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	182
—	2	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	10	183
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	184
1	—	—	—	—	1	—	—	—	—	—	—	1	1	3	—	—	—	—	—	—	—	22	1	—	4	40	185
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	3	186
1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	187
—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	3	188
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	189
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	190
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	—	1	5	191

	Diseases	Results of the																													
		Symptom-free								Symptom-free. Dead from intercurrent disease								Symptom-free, then recurrence. Retreated and later symptom-free or improved								Symptom-free, then recurrence. Retreated and later symptom-free or improved. Dead from intercurrent disease							
		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Digestive tract (continued):																														
192	Sarcoma palat. duræ op. rec.	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
193	Sarcoma palat. duræ op. *)	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
194	Tumor palat. mollis op. *)	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
195	Cancer palat. mollis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
196	Sarcoma palat. mollis op. rec.	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
197	Sarcoma palat. mollis op. *)	2	—	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
198	Cancer uvulæ	—	1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
199	Periostitis mandibulæ	—	—	—	1	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
200	Fistula mandibularis	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
201	Actinomycosis mandibulæ	—	1	—	1	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
202	Tumor mandibulæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
203	Tumor mandibulæ præop. treatment op. *)	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
204	Cancer mandibulæ	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
205	Cancer mandibulæ præop. treatment op. *)	—	—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
206	Cancer mandibulæ op. rec.	—	—	—	—	—	1	2	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
207	Cancer mandibulæ op. *)	1	—	—	—	—	—	1	2	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
208	Sarcoma mandibulæ	2	—	1	—	—	—	—	3	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
209	Sarcoma mandibulæ op. rec.	1	1	—	—	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
210	Sarcoma mandibulæ op. *)	2	1	—	—	—	—	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
211	Pyorrhœa alveol.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
212	Periostitis alveol.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
213	Tumor œsophagi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
214	Cancer œsophagi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
215	Cancer œsophagi op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
216	Cancer ventriculi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
217	Myxosarcoma ventriculi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
218	Sarcoma ventriculi op. *)	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
219	Tumor intestini	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
220	Actinomycosis intestini	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
221	Sarcoma intestini	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
222	Tbc. ileo cœcalis op. *)	—	—	1	1	—	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
223	Tumor ileo cœcalis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
224	Cancer coli op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
225	Cancer coli op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
226	Sarcoma coli op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
227	Tbc. cæci	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
228	Actinomycosis colli	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
229	Cancer cæci	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
230	Cancer cæci op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
231	Cancer cæci op. *)	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

Symptom-free, then recurrence. Retreated and later progression. Hopeless — Duration of cure: 1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Temporary improved. Then progression or dead: 1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	192
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	193
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	2	194
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	*1*	2	195
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	196
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	197
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	198
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	4	199
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	200
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	1	—	4	201
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	1	1	—	2	5	202
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	203
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	8	2	1	9	21	204
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	205
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	3	10	206
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	207
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	2	—	—	—	7	208
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	209
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	3	210
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	3	4	211
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	212
—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	—	—	—	—	—	—	2	—	—	2	4	213
—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	—	—	—	—	—	—	16	—	2	15	35	214
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	215
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	8	—	—	60	68	216
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	217
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	218
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	219
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	220
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	2	221
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	1	—	—	—	4	222
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	223
1	—	—	—	—	—	1	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	2	—	—	1	5	224
1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	225
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	226
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	227
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	228
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	1	1	—	4	229
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	1	—	—	3	230
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	231

	Diseases	Results of: Symptom-free — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Digestive tract (continued):																								
232	Tbc. recti	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
233	Ulcus recti præop. treatment op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
234	Polyposis recti	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
235	Cancer recti inop.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
236	Cancer recti præop. treatment op.*)	—	—	—	2	—	—	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
237	Cancer recti non rad. op.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
238	Cancer recti op. rec.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
239	Cancer recti op.*)	—	—	1	—	1	1	1	*4*	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	*1*
240	Sarcoma recti non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
241	Sarcoma recti op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
242	Sarcoma recti op.*)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
243	Varices hæmorrhoidales	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
244	Kraurosis ani	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
245	Tbc. ani	—	—	—	1	1	1	3	*6*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
246	Cancer ani	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
247	Actinomycosis hepatis op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
248	Cancer pancreatis inop.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
249	Sarcoma lymphogl. mesenterial.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
250	Tbc. peritonei	1	—	1	—	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
251	Myxoma peritonei	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
252	Cancer peritonei	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
253	Sarcoma peritonei	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
254	Tumor pelvis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
255	Tumor pelvis non rad. op.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
256	Cancer pelvis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
257	Sarcoma pelvis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
258	Sarcoma pelvis non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
259	Tumor retroperitonealis	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
260	Tumor retroperitonealis non rad. op.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
261	Sarcoma retroperitonealis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
262	Sarcoma retroperitonealis non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	*1*
263	Sarcoma retroperitonealis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
264	Sarcoma retroperitonealis op.*)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
265	Tumor abdominis	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment																																			
Symptom-free, then recurrence. Retreated and later progression. Hopeless								Improved								Temporary improved. Then progression or dead								Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																																			
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927							
—	1	—	—	—	—	*1*		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	232
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	233
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	234
—	1	—	—	—	—	*1*		—	—	—	—	—	—	3	*3*	7	—	—	—	—	—	7		23	*2*	*2*	*46*	83	235
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*6*	*1*	*1*	—	12	236
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	237
—	—	—	1	—	—	*1*		—	—	—	—	—	—	2	*2*	1	—	—	—	—	—	*1*		7	—	—	5	17	238
1	—	—	1	—	—	*1*		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*2*	—	*1*	—	9	239
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	240
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	*1*	—	—	1	241
—	—	—	—	—	—	—		—	—	—	—	—	—	1	*1*	1	—	—	—	—	—	*1*		*1*	—	—	—	1	242
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	*2*	6	243
—	—	—	—	—	—	—		—	—	—	—	1	1	—	*2*	1	—	—	—	—	—	*1*		—	*1*	—	—	10	244
—	1	—	—	—	—	*1*		—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*		—	—	*1*	—	4	245
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	246
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	247
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	248
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	249
—	—	—	1	—	—	*1*		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*3*	—	—	—	6	250
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	251
—	—	—	—	—	—	—		—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—		*2*	*1*	—	*1*	5	252
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*		—	—	—	—	1	253
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*6*	—	—	*1*	7	254
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	255
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	1	256
—	—	—	—	—	—	—		—	—	—	—	1	—	1	*2*	—	—	—	—	—	—	—		*5*	*1*	*1*	—	9	257
—	—	—	—	—	—	—		—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—		—	—	—	—	1	258
—	—	—	—	—	—	—		—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—		*6*	—	—	—	8	259
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		*1*	—	—	—	2	260
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	*1*	—	1	261
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	262
—	—	1	—	—	—	*1*		—	—	—	—	—	1	1	*2*	—	—	—	—	—	—	—		—	—	—	—	3	263
1	—	—	—	—	—	*1*		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		—	—	—	—	1	264
—	—	—	—	—	—	—		—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*		1	—	—	—	3	265

	Diseases	Results of the treatment: Symptom-free: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Bones and Joints:																								
266	Periostitis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
267	Osteitis fibrosa	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
268	Arthritis deformans	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
269	Omarthritis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
270	Arthritis urica	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
271	Osteitis et arthritis tbc.	1	2	2	2	—	1	—	*8*	1	1	—	—	—	—	—	*2*	—	—	—	—	—	—	—	—
272	Tumor benign.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
273	Tumor malign.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
274	Cancer	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
275	Sarcoma	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
276	Sarcoma non rad. op.	—	—	—	1	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
277	Sarcoma op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*
278	Sarcoma op. *)	2	—	—	—	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Kidneys and Urinary tract:																								
279	Hypernephroma renis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
280	Hypernephroma renis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
281	Hypernephroma renis op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
282	Fistula tbc. renis	1	—	1	—	—	—	—	*2*	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—
283	Sarcoma renis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
284	Sarcoma renis op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
285	Papilloma vesicæ urin	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
286	Papilloma vesicæ urin. op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
287	Tbc. vesicæ urin.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
288	Cancer vesicæ urin.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
289	Cancer vesicæ urin. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
290	Cancer vesicæ urin. op. *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
291	Sarcoma vesicæ urin.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
292	Sarcoma vesicæ urin. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
293	Polyposis urethræ op. rec.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
294	Cancer urethræ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Male genital organs:																								
295	Tumor testis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
296	Tumor malign. testis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
297	Tumor malign. testis op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
298	Cancer testis op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
299	Cancer testis op. *)	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment																																						
Symptom-free, then recurrence. Retreated and later progression. Hopeless								Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																																						
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927		Adm. in 1921		Adm. in 1922		Adm. in 1923		Adm. in 1924		Adm. in 1925		Adm. in 1926		Adm. in 1927		Total 1921—1927		1 Year		2 Years		3 Years		4 Years		5 Years		6 Years		Total 1921—1927																		

	Diseases	Results of: Symptom-free — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Male genital organs (continued):																								
300	Sarcoma testis op. rec.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
301	Sarcoma testis præop. treatm. op.*)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
302	Sarcoma testis op. *)	—	—	—	—	1	—	1	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
303	Epididymitis tbc.	1	—	—	1	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
304	Epididymitis tbc. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
305	Tumor malign. epididymis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
306	Sarcoma epididymis op. *)	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
307	Hypertrophia prostatæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
308	Tumor malign. prostatæ non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
309	Cancer prostatæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
310	Cancer prostatæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
311	Cancer prostatæ op. *)	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
312	Sarcoma prostatæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
313	Sarcoma prostatæ non rad. op.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
314	Tumor benign. penis	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
315	Induratio plast. penis	—	—	—	1	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
316	Cancer penis	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
317	Cancer penis op. rec.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
318	Cancer penis præop. treatm. op.	—	—	—	1	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
319	Cancer penis op. *)	—	1	—	—	—	1	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
320	Tumor scroti	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—
	Female genital organs:																								
321	Eczema vulvæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
322	Ulcus vulvæ	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
323	Pruritus vulvæ	—	1	—	—	2	2	—	*5*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
324	Kraurosis vulvæ	—	1	—	—	1	5	1	*8*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
325	Tumor benign. vulvæ	—	—	—	—	2	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
326	Cancer vulvæ	1	2	2	2	2	6	4	*19*	—	—	1	—	—	1	—	*2*	—	—	—	—	—	—	—	—
327	Cancer vulvæ non. rad op.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
328	Cancer vulvæ op. rec.	—	—	—	—	1	1	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
329	Cancer vulvæ op. *)	1	1	—	—	—	—	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
330	Sarcoma vulvæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
331	Sarcoma vulvæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
332	Ulcus vaginæ	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
333	Cancer vaginæ	1	—	1	—	—	1	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
334	Sarcoma vaginæ	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
335	Adenomyecitis rectovaginale	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																											
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	3	—	—	—	5	300
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	301
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	3	302
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	303
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	304
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	1	305
—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	—	—	—	—	—	—	—	—	2	—	1	306
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	2	—	—	—	—	—	2	1	1	—	—	4	307
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	—	—	—	—	—	1	9	2	1	4	19	308
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	—	—	—	—	—	—	2	—	—	—	3	309
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	310
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	1	311
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	—	—	—	1	3	314
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	6	315
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	1	1	—	—	—	—	2	5	—	1	2	12	316
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	1	—	—	—	3	317
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	318
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	319
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	320
1	—	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	321
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	322
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	4	3	12	323
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	4	14	324
5	1	—	1	—	—	7	—	—	—	—	—	1	3	4	5	—	—	—	—	—	5	23	1	7	9	77	326
1	—	—	1	—	—	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	3	327
1	1	—	—	1	—	3	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6	1	1	5	18	328
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	8	329
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	330
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	331
2	—	—	—	—	—	2	—	—	—	—	—	1	—	1	1	—	—	—	—	—	1	12	—	1	10	29	333
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	334
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	335

	Diseases	Results of the treatment: Symptom-free: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Female genital organs (continued):																								
336	Cancer colli uteri [1])	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
337	Cancer colli uteri op. rec.	—	1	—	3	3	2	1	*10*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
338	Cancer colli uteri op. *)	—	2	1	2	2	4	—	*11*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
339	Chorion epithelioma corp. uteri op. rec.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
340	Cancer corporis uteri [2])	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
341	Cancer corporis uteri non rad. op.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
342	Cancer corporis uteri op. rec.	1	2	2	4	—	—	—	*9*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
343	Cancer corporis uteri op. *)	1	1	4	6	8	7	5	*32*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
344	Sarcoma uteri	—	—	2	—	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
345	Sarcoma uteri op. rec.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
346	Sarcoma uteri op. *)	—	—	—	1	—	2	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
347	Cancer tubæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
348	Cancer tubæ op. *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
349	Tumor ovarii	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
350	Tumor ovarii op. rec.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
351	Tumor ovarii op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
352	Cancer ovarii inop.	—	1	—	—	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
353	Cancer ovarii non rad. op.	—	2	—	—	2	1	2	*7*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
354	Cancer ovarii op. rec.	1	3	—	1	—	—	—	*5*	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	*2*
355	Cancer ovarii op. *)	2	1	6	7	2	4	—	*22*	—	—	—	—	—	1	—	*1*	—	—	—	—	8	1	—	*9*
356	Sarcoma ovarii op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
357	Sarcoma ovarii op. *)	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
358	Metropathia	17	17	23	23	18	16	5	*119*	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—
359	Myoma	22	21	27	27	14	16	10	*137*	1	—	2	—	—	—	—	*3*	—	—	1	—	—	—	—	*1*
360	Dysmenorrhœ	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*
361	Salpingitis chronica	—	1	1	—	—	—	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
362	Salpingitis tbc.	1	1	2	5	1	2	—	*12*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
363	Varia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Tendons and Bursæ:																								
364	Tendovaginitis tbc.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
365	Bursitis tbc.	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
366	Sarcoma tend. op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
367	Sarcoma tend. op. *)	—	—	1	1	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
368	Ganglion	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
369	Ganglion op. rec. op. *)	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
370	Peritendinitis chron.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
371	Contractura Dupuytren.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

[1]) See: Table III. — *) Prophylactically treated. — [2]) See: Table IV.

| the treatment | | |

	Diseases	Results of the																										
		Symptom-free								Symptom-free. Dead from intercurrent disease								Symptom-free, then recurrence. Retreated and later symptom-free or improved											
		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	
	Connective tissue and Fasciæ:																											
372	Tumor benign.	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
373	Tumor benign. op. *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
374	Tumor malign.	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	2	—	
375	Tumor malign. op. rec.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
376	Tumor malign. op. *)	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	*1*
377	Cancer non rad. op.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
378	Sarcoma	—	1	2	—	—	—	—	*3*	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	1	—	
379	Sarcoma non rad. op.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
380	Sarcoma præop. treatm. op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	
381	Sarcoma op. rec.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
382	Sarcoma op. *)	2	4	2	2	1	—	8	*19*	1	—	—	—	—	—	—	*1*	—	2	—	—	—	2	—	*4*	4	—	
383	Myxoma	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
384	Myxoma op. *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
	Skin:																											
385	Ulcus cutis	—	1	2	3	2	1	1	*10*	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	
386	Furunculosis	4	1	6	3	2	—	1	*17*	—	—	—	—	—	—	—	—	—	2	—	—	—	—	—	*2*	—	—	
387	Folliculitis	—	1	4	3	2	—	—	*10*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*	—	—	
388	Eczema	4	10	3	9	8	2	10	*46*	—	—	—	—	—	—	—	—	1	1	1	1	—	—	—	*4*	—	—	
389	Dermatitis subchron.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
390	Pemphigus	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
391	Morbus Bowen	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
392	Herpes	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
393	Lupus erythematosus	2	1	—	—	—	—	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
394	Rhinophyma	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
395	Lupus pernio	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
396	Lupus vulgaris	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
397	Ichtyosis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
398	Erythema indurativum	1	—	1	—	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
399	Seborrhoea	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
400	Psoriasis	1	—	—	—	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
401	Pruritus	1	—	—	2	3	—	3	*9*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*	—	—	
402	Infiltratio cutis	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
403	Ulcus anticruris	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	
404	Tbc. cutis	5	2	7	3	3	1	—	*21*	—	1	—	—	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	

*) Prophylactically treated.

| the treatment | | |

	Diseases	Results of: Symptom-free								Symptom-free. Dead from intercurrent disease							Symptom-free, then recurrence. Retreated and later symptom-free or improved						
		Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Adm. in 1921	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Skin (continued):																						
405	Atheroma	—	1	—	—	—	—	2	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
406	Actinomycosis cutis	—	—	—	—	1	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
407	Fibroma cutis	—	2	1	1	1	1	—	*6*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
408	Dermoides	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
409	Nævus pigm. et pil.	5	5	3	1	—	3	4	*21*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
410	Xanthelasma	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
411	Cheloides	3	3	6	6	4	13	5	*40*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
412	Lipoma	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
413	Papilloma	1	1	1	1	1	2	2	*9*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
414	Verrucæ	75	107	115	197	184	245	186	*1,109*	—	—	—	—	—	4	—	*4*	—	1	3	4	5	2	— *15*
415	Clavus	3	—	—	—	1	6	3	*13*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
416	Hyperkeratosis	—	—	1	1	—	1	1	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
417	Cancer cutis	36	48	34	49	66	86	46	*365*	12	12	6	9	9	5	1	*54*	4	3	3	2	3	4	— *19*
418	Cancer cutis op. rec.	1	4	3	2	2	9	5	*26*	—	1	—	—	—	—	—	*1*	—	1	1	—	—	2	1 *5*
419	Cancer cutis op. *)	—	2	1	—	1	2	2	*8*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	— *1*
420	Melanosarcoma cutis	—	—	1	1	—	1	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
421	Melanosarcoma cutis op. rec.	—	1	1	—	—	1	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
422	Melanosarcoma cutis op. *)	—	—	—	—	1	1	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
423	Sarcoma cutis	1	4	1	—	—	—	—	*6*	—	—	1	1	—	—	—	*2*	—	—	—	—	—	—	— —
424	Sarcoma cutis non rad. op.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
425	Sarcoma cutis op. rec.	1	2	—	1	—	1	—	*5*	—	1	—	—	—	—	—	*1*	—	—	—	1	—	—	— *1*
426	Sarcoma cutis op. *)	1	3	2	1	—	2	2	*11*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
	Hair and Nails:																							
427	Paronychia	—	—	2	2	2	3	2	*11*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
428	Alopecia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
429	Onychogryphosis	1	1	—	—	—	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
430	Onychomycosis	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
431	Sycosis parasitaria	2	1	—	1	1	—	—	*5*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
	Sebaceous- and Sweatglands:																							
432	Hidradenitis	44	41	15	13	19	11	13	*156*	—	—	—	—	—	—	—	—	1	2	—	—	—	—	— *3*
433	Hyperhidrosis	2	3	1	—	—	—	—	*6*	—	—	—	—	—	—	—	—	1	—	—	—	—	—	— *1*
434	Trichiasis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
	Eye:																							
435	Trachoma	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
436	Chalazion	—	1	—	—	—	—	1	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
437	Granuloma conjunct. et palp.	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —
438	Cancer palpebræ	3	5	14	12	14	20	15	*83*	5	4	6	4	2	—	—	*21*	1	—	—	—	1	—	— *2*
439	Cancer palpebræ op. rec.	—	1	—	4	2	1	—	*8*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	— —

*) Prophylactically treated.

| the treatment | | |

	Diseases	Results of: Symptom-free — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Eye (continued):																								
440	Cancer palpebræ op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—
441	Sarcoma palpebræ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
442	Tbc. gland. lacrimalis	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
443	Cancer sacc. lacrim. non rad. op.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
444	Dacryocystitis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
445	Conjunctivitis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
446	Tumor conjunctivæ	—	—	—	1	1	—	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
447	Cancer conjunctivæ	—	—	2	1	—	—	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
448	Sarcoma conjunctivæ	2	—	1	—	1	—	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
449	Tumor orbitæ	2	—	—	—	2	1	—	*5*	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	*1*
450	Tumor orbitæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
451	Tumor orbitæ op. *)	—	—	—	—	1	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
452	Sarcoma orbitæ	—	—	—	—	1	2	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
453	Sarcoma orbitæ op. rec.	—	—	—	—	1	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
454	Sarcoma orbitæ op. *)	—	—	—	1	1	2	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
455	Cancer cornæ	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
456	Sarcoma cornæ	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Ear:																								
457	Ulcus chron. auriculi	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
458	Tumor malign. auriculi	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
459	Tumor malign. auriculi op. rec.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
460	Cancer auriculi	—	3	2	4	2	4	—	*15*	1	1	1	3	—	—	—	*6*	—	—	—	—	—	1	—	*1*
461	Cancer auriculi op. rec.	—	—	—	—	—	—	3	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
462	Cancer auriculi op.*)	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
463	Sarcoma auriculi op. rec.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
464	Otoscleros	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
465	Otitis media chron.	—	—	—	1	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
466	Polyposis meatus aucust.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
467	Cancer meatus aucust.	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Thyroid:																								
468	Hyperplasia thymii	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
469	Strumitis	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
470	Tumor thyreoideæ	—	—	1	—	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
471	Struma	4	3	6	1	2	2	—	*18*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
472	Hyperthyreoidism.	8	5	5	2	3	2	1	*26*	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	*1*

*) Prophylactically treated.

the treatment

Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																											
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	2	440
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	441
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	442
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	443
—	—	—	—	—	—	—	—	—	1	1	1	—	1	4	—	—	—	—	—	—	—	1	—	—	—	1	444
—	—	—	—	—	—	—	—	—	—	—	—	—	2	2	—	—	—	—	—	—	—	1	1	1	—	6	445
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	7	446
—	—	—	—	—	—	—	—	2	—	—	1	—	2	5	3	—	—	—	—	—	3	1	—	1	—	3	447
1	—	—	—	—	—	—	—	—	—	—	1	1	2	4	—	—	—	—	—	—	—	2	2	—	1	14	448
—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—	—	—	—	—	—	1	—	—	—	15	449
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	1	—	—	—	—	—	1	2	—	—	—	2	450
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	451
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	1	—	—	—	—	—	1	2	—	—	—	7	452
—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	3	—	1	—	6	453
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	454
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	455
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	2	456
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	1	3	457
—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	—	—	—	—	—	—	—	—	—	1	458
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	459
1	—	—	—	—	—	—	—	—	—	—	—	—	3	3	—	—	—	—	—	—	—	1	—	2	1	29	460
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	5	461
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	462
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	463
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	2	464
—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	—	—	—	—	—	—	—	2	1	1	1	8	465
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	466
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	4	—	2	1	8	467
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	468
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	2	469
—	—	—	—	—	—	—	—	—	—	—	1	—	1	2	—	—	—	—	—	—	—	1	—	—	—	5	470
1	—	—	—	—	—	—	—	1	1	1	3	1	2	8	—	—	—	—	—	—	—	3	1	25	349	404	471
								1	1	1	4	4	6	16								2	4	16	13	78	472

	Diseases	Results of the treatment: Symptom-free: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved: Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Thyroid (continued):																								
473	Morbus Basedowi	7	5	4	6	3	1	—	*26*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
474	Morbus Basedowi op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
475	Struma maligna	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
476	Struma maligna non rad. op.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
477	Struma maligna op. rec.	1	1	—	1	—	—	—	*3*	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—
478	Struma maligna op. *)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
479	Cancer thyreoideæ	—	—	—	—	1	—	—	*1*	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—
480	Cancer thyreoideæ non rad. op.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
481	Cancer thyreoideæ op. rec.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
482	Cancer thyreoideæ op. *)	1	—	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
483	Sarcoma thyreoideæ non rad. op	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
484	Sarcoma thyreoideæ op. *)	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Hypophysis cerebri:																								
485	Tumor hypophyseos	2	—	—	1	1	—	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Brain and Nervous system:																								
486	Tumor cerebri	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
487	Tumor cerebri non rad. op.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*
488	Tumor cerebri op. *)	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
489	Sarcoma duræ non rad. op.	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
490	Tumor extra duralis non rad. op.	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
491	Glioma medullæ spinalis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
492	Tumor medullæ spinalis	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
493	Tumor medullæ spinalis op. *)	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
494	Sarcoma medullæ spinalis	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
495	Syringomyelia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
496	Chordoma maligna non rad. op.	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
497	Paralysis bulbaris	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
498	Elephantiasis neuromatosa	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
499	Neuralgia	—	—	—	2	—	—	1	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
500	Sclerosis en plaque	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Breast:																								
501	Mastitis chronica	2	—	1	—	—	—	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*
502	Mastitis chron. op rec.	—	1	—	—	—	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
503	Mastitis chron. præop. treatm.	—	—	—	—	—	2	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
504	Mastitis chron. op *)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
505	Mastitis hæmorrhagia	—	—	—	—	1	1	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

*) Prophylactically treated.

the treatment																																
Symptom-free, then recurrences. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921 – 1927	
Duration of cure																																
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927						
1	—	—	—	—	—	*1*	2	—	1	2	3	3	8	*19*	2	1	—	—	—	—	*3*	*2*	*2*	*23*	*25*	101	473
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	*3*	4	474
—	—	—	—	—	—	—	—	—	—	—	—	2	7	*10*	1	—	—	—	—	—	*1*	*14*	*1*	—	*4*	30	475
—	—	—	—	—	—	—	—	—	—	1	—	—	1	*1*	—	—	—	—	—	—	—	*1*	—	—	—	3	476
—	—	1	—	—	—	*1*	—	—	—	—	—	—	1	*1*	2	—	—	—	—	—	*2*	—	—	—	—	7	477
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	*1*	—	3	478
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*	*1*	—	—	—	4	479
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	480
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*2*	—	—	—	2	481
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	482
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	1	483
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	484
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*5*	*1*	*5*	*3*	18	485
1	—	—	—	—	—	*1*	—	—	—	—	—	2	—	*2*	1	—	—	—	—	—	*1*	*2*	—	*1*	*1*	8	486
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*2*	—	—	—	—	—	—	—	*2*	—	—	—	5	487
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	488
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	—	1	489
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	1	490
—	—	—	—	—	—	—	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	1	491
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*2*	—	*2*	—	4	492
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	2	493
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	494
—	—	—	—	—	—	—	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	—	—	1	495
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	496
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	1	497
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	—	—	1	498
—	—	—	—	—	—	—	—	—	—	1	1	—	—	*2*	1	—	—	—	—	—	*1*	*5*	—	*11*	*6*	28	499
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	1	500
—	—	—	—	—	—	—	—	—	—	—	—	—	1	*1*	—	—	—	—	—	—	—	—	—	*1*	*9*	15	501
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	502
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	*1*	—	—	2	503
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	504
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	505

	Diseases	Results of: Symptom-free — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free. Dead from intercurrent disease — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	Symptom-free, then recurrence. Retreated and later symptom-free or improved — Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927
	Breast (continued):																								
506	Mastitis serosanguinolenta præop. treatment	—	—	—	—	1	2	1	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
507	Tumor mammæ	1	—	—	—	2	—	—	*3*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
508	Tumor mam. præop. treatment op. *)	—	4	4	3	5	2	1	*19*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
509	Tumor mam. op. *)	—	—	—	—	2	2	—	*4*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
510	Cancer mammæ	2	1	1	—	5	4	1	*14*	—	—	1	—	—	—	—	*1*	—	—	1	—	—	—	—	*1*
511	Cancer mammæ inop.	—	—	—	—	—	1	—	*1*	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	*1*
512	Cancer mammæ op. rec.	—	1	1	2	3	6	3	*16*	—	—	—	—	—	—	—	—	—	1	—	1	1	1	—	*4*
513	Cancer mammæ præop. treatment op. *)	1	9	10	18	11	13	25	*87*	—	1	—	—	—	—	—	*1*	—	—	3	—	1	2	—	*6*
514	Cancer mammæ op.	15	10	3	10	18	27	11	*94*	4	2	1	2	3	1	1	*14*	1	3	3	—	3	5	—	*15*
515	Sarcoma mammæ	—	—	—	1	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
516	Sarcoma mammæ op. rec.	—	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
517	Sarcoma mammæ præop. treatment op.*)	—	—	1	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
518	Sarcoma mammæ op. *)	—	—	1	—	1	—	—	*2*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
519	Varia	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Total	—	—	—	—	—	—	—	**4,100**	—	—	—	—	—	—	—	**160**	—	—	—	—	—	—	—	**139**

*) Prophylactically treated.

t h e t r e a t m e n t

Symptom-free, then recurrence. Retreated and later progression. Hopeless							Improved								Temporary improved. Then progression or dead							Not improved	Recently treated	Abandoned treatment	Not suitable for treatment	Number of cases controlled 1921—1927	
Duration of cure																											
1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927	Adm. in 1921	Adm. in 1922	Adm. in 1923	Adm. in 1924	Adm. in 1925	Adm. in 1926	Adm. in 1927	Total 1921—1927	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years	Total 1921—1927							
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	5	506
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*2*	—	*44*	49	507
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	20	508
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	5	509
1	3	5	—	1	—	—	*9*	—	—	—	—	—	1	3	*4*	6	2	—	—	—	—	*8*	*24*	*1*	*6*	*45*	113	510
1	—	—	—	—	—	—	—	—	—	—	—	1	—	1	*2*	5	1	—	—	—	—	*6*	*31*	—	*2*	*24*	67	511
4	14	3	—	—	—	—	*17*	—	—	—	—	2	1	8	*11*	14	1	—	—	—	—	*15*	*86*	*6*	*12*	*37*	204	512
6	14	10	3	2	1	—	*30*	—	—	—	—	—	1	1	*2*	5	2	—	—	—	—	*7*	*16*	*7*	*13*	—	169	513
15	32	16	3	2	—	—	*53*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*35*	*6*	*17*	*13*	247	514
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	2	515
	—	—	—	—	—	—	—	—	—	—	—	—	1	1	*1*	—	—	—	—	—	—	—	—	—	—	—	2	516
	1	—	—	—	—	—	*1*	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	517
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1*	—	—	—	3	518
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	*1,777*	1,777	519
	—	—	—	—	—	—	**236**	—	—	—	—	—	—	—	**734**	—	—	—	—	—	—	**209**	**1,225**	**251**	**906**	**5,592**	**14,608**	

Table II. Cases treated with electroendothermy and desiccation at Radiumhemmet in Stockholm 1927.

Diseases	Symptom-free	Improved	Temporary improved	Recently treated	Died	Total	Also radiological treatment	No radiological treatment
Blood and Bloodgenerating organs:								
Lymphosarcoma op. rec.	1	—	—	—	—	1	1	—
Teleangiectasie	1	—	—	1	—	2	—	2
Hæmangioma vinosum	11	5	—	1	—	17	3	14
Hæmangioma cavernosum	8	2	—	1	—	11	1	10
Nose and Throat:								
Tumor cavitatis nasi	—	—	—	1	—	1	1	—
Tumor tonsillæ	1	—	—	—	—	1	1	—
Tbc. pharyngis	—	1	—	—	—	1	1	—
Digestive tract:								
Leucoplaciæ	4	—	—	1	—	5	—	5
Ulcus labii	4	—	—	1	—	5	—	5
Tumor labii	3	—	—	—	—	3	—	3
Cancer labii	4	1	—	—	1	6	5	1
Tumor buccæ	2	—	—	—	—	2	—	2
Cancer buccæ	2	2	—	—	1	5	5	—
Cancer buccæ op. rec.	—	1	1	—	1	3	3	—
Ulcus linguæ	1	—	—	1	—	2	2	—
Papilloma linguæ	1	—	—	1	—	2	—	2
Tumor linguæ	2	—	—	—	—	2	1	1
Cancer linguæ	—	1	—	1	2	4	4	—
Tymor reg. subling	1	—	—	—	—	1	1	—
Epulis	1	—	—	—	—	1	1	—
Cancer gingivæ	—	—	—	—	1	1	1	—
Cancer recti op. rec.	—	1	—	—	—	1	1	—
Fistula ad anum	1	—	—	—	—	1	1	—
Urinary tract:								
Polyposis urethræ	1	—	—	—	—	1	—	1
Male genital organs:								
Cancer penis	—	1	—	—	—	1	1	—

Diseases	Symptom-free	Improved	Temporary improved	Recently treated	Died	Total	Also radiological treatment	No radiological treatment
Female genital organs:								
Kraurosis vulvæ	1	—	—	—	—	1	1	—
Tumor vulvæ	1	—	—	—	—	1	—	1
Cancer vulvæ	5	2	3	—	—	10	10	—
Cancer vulvæ op. rec.	1	—	2	1	—	4	4	—
Connective tissue:								
Sarcoma op. rec.	—	1	—	—	—	1	1	—
Skin:								
Ulcus chronic	4	—	—	1	—	5	—	5
Eczema	—	1	—	—	—	1	—	1
Atheroma	1	—	—	—	—	1	—	1
Fibroma	49	7	—	5	—	61	—	61
Nævus pigm.	21	7	—	3	—	31	2	29
Xanthelasma	1	—	—	—	—	1	—	1
Cheloides	—	4	—	—	—	4	4	—
Verrucæ	102	20	3	5	—	130	48	82
Corn cutan	4	—	—	—	—	4	—	4
Cancer cutis	7	4	—	3	—	14	9	5
Cancer cutis op. rec.	3	—	—	—	—	3	3	—
Sarcoma cutis	2	—	—	1	—	3	2	1
Eye:								
Chalazion palpebræ	1	—	—	—	—	1	—	1
Breast:								
Cancer mammæ	5	2	2	1	—	10	10	—
Cancer mammæ op. rec.	1	1	3	—	—	5	5	—
Sarcoma mammæ op. rec.	—	1	—	—	—	1	1	—
Total	258	65	14	29	6	372	134	238
							372	

Table III. Result of the treatment in all cases of cancer colli uteri treated 1914—1927.

Year	Cases	Symptomfree after																									
		1/2 yr	%	3/4 yr	%	1 yr	%	1.5 yr	%	2 yr	%	3 yr	%	4 yr	%	5 yr	%	6 yr	%	7 yr	%	8 yr	%	9 yr	%	10 yr	%
1914...	26	14	53.8	14	53.8	12	46.2	9	34.6	9	34.6	8	30.8	7	26.9	7	26.9	5	19.2	5	19.2	5	19.2	5	19.0	4	15.4
1915...	40	22	55.0	21	52.5	15	37.5	15	37.5	14	35.0	13	32.5	13	32.5	13	32.5	12	30.0	10	25.0	10	25.0	10	25.0	9	22.5
1916...	47	15	31.9	10	21.3	8	17.0	6	12.8	4	8.5	4	8.5	4	8.5	4	8.5	3	6.4	3	6.4	1	2.1	1	2.1	1	2.1
1917...	63	34	53.9	23	36.5	20	31.7	14	22.2	13	20.6	10	15.9	10	15.9	9	14.3	8	12.7	8	12.7	8	12.7	8	12.7	7	11.1
1918...	41	21	51.2	21	51.2	19	46.3	17	41.5	15	36.6	13	31.7	12	29.3	11	26.8	10	24.4	10	24.4	10	24.4	10	24.4		
1919...	76	50	65.8	41	53.9	29	38.2	25	32.9	25	32.9	24	31.6	21	27.6	20	26.3	20	26.3	17	22.4	17	22.4				
1920...	96	53	55.2	48	50.0	40	41.7	32	33.3	31	32.3	29	30.2	28	29.2	26	27.1	25	26.0	24	25.0						
1921...	113	70	61.9	60	53.1	54	47.8	46	40.7	44	38.9	36	31.9	30	26.5	27	23.9	27	23.9								
1922...	132	61	46.2	54	40.9	50	37.9	43	32.6	40	30.3	34	25.8	29	22.0	28	20.4										
1923...	104	55	52.9	49	47.1	43	41.3	36	34.6	32	30.8	28	26.9	25	24.0												
1924...	148	86	58.1	75	50.7	69	46.6	58	39.2	50	33.8	44	29.7														
1925...	132	71	53.8	55	41.7	45	34.1	28	21.2	28	21.2																
1926...	140	80	57.1	69	49.3	61	43.6																				
1927...	149																										

Table IV. Result of the treatment in all cases of cancer corporis uteri 1913—1927.

Year	Cases	Symptomfree after																									
		1/2 yr	%	3/4 yr	%	1 yr	%	1.5 yr	%	2 yr	%	3 yr	%	4 yr	%	5 yr	%	6 yr	%	7 yr	%	8 yr	%	9 yr	%	10 yr	%
1913...	1	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	—		—		—		—		—		—		—	
1914...	1	1	100.0	1	100.0	—		—		—		—		—		—		—		—		—		—		—	
1915...	1	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	1	100.0	—		—		—	
1916...	4	3	75.0	3	75.0	3	75.0	3	75.0	3	75.0	2	50.0	2	50.0	2	50.0	2	50.0	2	50.0	2	50.0	1	25.0	1	25.0
1917	5	4	80.0	4	80.0	4	80.0	4	80.0	4	80.0	4	80.0	4	80.0	4	80.0	3	60.0	3	60.0	1	20.0	1	20.0	1	20.0
1918...	7	6	85.7	5	71.4	5	71.4	3	42.9	3	42.9	3	42.9	3	42.9	3	42.9	3	42.9	3	42.9	3	42.9	3	42.9		
1919...	3	3	100.0	3	100.0	2	66.7	2	66.7	2	66.7	2	66.7	2	66.7	2	66.7	2	66.7	2	66.7	2	66.7				
1920...	12	11	91.7	11	91.7	11	91.7	9	75.0	9	75.0	8	66.7	7	58.3	5	41.7	4	33.3	4	33.3						
1921...	12	8	66.7	8	66.7	8	66.7	8	66.7	8	66.7	7	58.3	4	33.3	4	33.3	4	33.3								
1922...	23	16	69.6	13	56.5	11	47.8	11	47.8	11	47.8	11	47.8	9	39.1	7	30.4										
1923...	16	14	87.5	14	87.5	14	87.5	10	62.5	9	56.3	9	56.3	9	56.3												
1924...	15	10	66.7	8	53.3	8	53.3	7	46.7	7	46.7	6	40.0														
1925...	18	16	88.9	14	77.8	14	77.8	13	72.2	13	72.2																
1926...	37	26	70.3	26	70.3	25	67.6																				
1927...	19																										

Stockholm, K. L. Beckmans Boktr., 1928.